JOURNAL
of the
American Veterinary Medical Association
Formerly AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

Vol. LXXXVII, N. S. Vol. 40 DECEMBER, 1935 No. 6

MICHIGAN TAKES IMPORTANT STEP IN PUBLIC HEALTH WORK

In another section of this issue of the JOURNAL is published a brief report of a postgraduate short course in milk and meat inspection given by the Division of Veterinary Science of Michigan State College for the particular benefit of the veterinarians of seven counties in the southwestern part of the state. The short course was arranged and conducted at the specific request of the W. K. Kellogg Foundation, in connection with the Michigan Community Health Project being undertaken by that organization.

Although the present Michigan requirements do not include a veterinarian in the setup of a county health department, it is beginning to be recognized that no program for community health can be complete unless the veterinarian is included. A great deal has been said and written on this subject during the past quarter of a century, but entirely too small a part of this more or less academic discussion has been translated into action. Some years ago, New Jersey took an advance step in recognizing the value of veterinary service in providing that a veterinarian should be included in the personnel of the State Department of Health. In

other states, from time to time, veterinarians have acted as consultants to boards of health, and in a few of the large cities veterinarians have been members of municipal health bodies. But these cases have been entirely too few, and it is believed that an impartial investigation would disclose that in a majority of the cases in which this recognition has been extended to the veterinary profession, it has been largely because of the prominence of some individual veterinarian, rather than the recognition and acceptance of any basic principle in public health work.

The Michigan Community Health Project definitely recognizes the veterinarian as a necessary participant in any modern, comprehensive, forward-looking health program. The education and training of the veterinarian of today admirably equip him to play an important rôle in any health project, county, state or national in scope. He possesses a knowledge of the diseases of animals that are communicable to man that is not a part of the armamentarium of the man of any other training. The veterinarian is prepared to put this knowledge to practical use by way of safeguarding our food supplies, particularly meat and milk. This is something that is needed in our rural communities right now to even a greater degree than is the case in most of our large urban centers.

EXECUTIVE BOARD ELECTION

Members of the A. V. M. A. located in Executive Board District 8 (Kansas, Missouri, Oklahoma, Arkansas, Texas and Louisiana) are now casting their ballots for the purpose of determining who shall represent them on the Executive Board for the balance of the current year. This is a special election to select a successor to Dr. J. C. Flynn, who resigned as member on the Board following his election to the presidency at Oklahoma City. The primary election is over and ballots for the election proper have been mailed to all members in District 8, in good standing. More than 60 per cent of the ballots already have been returned.

In looking over the names of the five nominees in the present election, we are impressed by several facts. First of all, each of the five candidates may be said to be in a different branch of veterinary medicine. One is a Bureau of Animal Industry inspector, one a practitioner, one a college professor, one a state veterinarian and the other is a commercial veterinarian.

Then, upon comparing the candidates in this election with those whose names appeared on the ballot in the election held in the same district in 1932, we find that there is not a single duplica-

tion. In other words, the five men now being voted on are an entirely different five from the ones who were candidates in 1932. (This was the special election held to select a successor to Dr. N. F. Williams, when the latter was elected president at the Atlanta meeting.) In the 1932 election, the five candidates selected in the primary were an entirely different five from those who constituted the ticket in the regular election in 1931. This means that in three elections held in 1931, 1932 and 1935, in District 8, 15 different names have appeared on the three ballots. This is in marked contrast to elections held in some of the other Executive Board districts in recent years. For example, in the special election in District 4, held earlier this year to select a successor to the late Dr. C. A. Cary, all of the five nominees in the special election had been nominated in the regular election held in 1934.

The nominees in the special election now being held in District 8 are as follows:

ALLEN, L. J. — Oklahoma City, Okla.

Inspector-in-charge, U. S. Bureau of Animal Industry. Graduate of Ontario Veterinary College, 1895. Joined A. V. M. A., 1918. Resident secretary for Oklahoma, 1919-21.

BOWER, C. W. — Topeka, Kan.

Practitioner. Graduate of Kansas State College, 1918. Joined A. V. M. A., 1918. Resident secretary for Kansas, 1927-28 and 1933-34. Chairman of Section on Small Animals, 1928-30.

FRANK, E. R. — Manhattan, Kan.

Professor of Surgery and Large Animal Clinics, Kansas State College. Graduate of Kansas State College, 1924. Joined A. V. M. A., 1926.

HISEL, C. C. — Oklahoma City, Okla.

State Veterinarian of Oklahoma. Graduate of Kansas City Veterinary College, 1916. Joined A. V. M. A., 1930. Resident secretary for Oklahoma, 1931-32. Chairman of Committee on Local Arrangements, 1934-35.

LOCKHART, ASHE — Kansas City, Mo.

President, Ashe Lockhart, Inc. Graduate of Kansas City Veterinary College, 1915. Joined A. V. M. A., 1916. Member of Committee on Audit, 1921-22. Chairman of Committee on Veterinary Biological Products, 1924-25. Member of Special Committee on Distemper, 1926-27 and 1930-32. Resident secretary for Missouri, 1933-.

THIRTY-ONE STATES NOW ACCREDITED

The tuberculin testing of cattle in Tennessee was completed early in October and the state was designated by the U. S. Department of Agriculture as a modified accredited area, the 26th to be inscribed on the honor roll of tuberculosis-free states.

Under date of November 7, announcement was made of the completion of the work in five more states: Massachusetts, South

Carolina, Georgia, Alabama and Louisiana. This group of five states represents the largest increment to the list ever announced at one time. The number of states now practically free from bovine tuberculosis is 31, just one short of two-thirds of all the states.

Counties that are now classed as modified accredited areas constitute approximately 88 per cent of all of the counties in the United States, according to the latest computation made by the U. S. Bureau of Animal Industry. Area work is in progress also in about 270 other counties. The "blackest" spots on the "TB" map are now located in California, South Dakota, Iowa, New York, Pennsylvania, New Jersey and Connecticut.

APOLOGIES, WASHINGTON

In the report on the enrollment of veterinary students, published in the November issue of the JOURNAL, the State College of Washington was erroneously included in the list of institutions which had not yet put into effect the requirement of a year of pre-veterinary work of all students matriculating in the College of Veterinary Medicine. The new requirement went into effect at Pullman this fall. The report on the number of veterinary students at the State College of Washington, as furnished by Dean Wegner, made no mention of pre-veterinary students, and this, to some extent, contributed to the error. Dean Wegner states that students taking pre-veterinary work at Pullman are not classified as veterinary students. In this respect, the situation is substantially the same as at Cornell University. Dean Wegner points out that it is difficult to determine accurately the number of so-called pre-veterinary students, although he has knowledge of some 68 students on the campus at Pullman who have expressed an interest in qualifying to enter the College of Veterinary Medicine. We regret the error in omitting Washington from the list of institutions that have adopted the requirement of a year of pre-veterinary work.

In any consideration of the number of students taking pre-veterinary studies, it should be kept in mind that there probably are many such students in colleges and universities not offering a course leading to a degree in veterinary medicine. In other words, it is not necessary that pre-veterinary studies be taken in a veterinary college. As a result of this fact, there is no convenient way at the present time of accurately determining the number of so-called pre-veterinary students throughout the country.

APPLICATIONS FOR MEMBERSHIP

(See July, 1935, JOURNAL)

FIRST LISTING

ALFREDSON, BERNARD V. 1123 Cleo St., Lansing, Mich.
D. V. M., Michigan State College, 1931
Vouchers: Lloyd B. Sholl and B. J. Killham.

BELLIS, WILLIAM C. Fall River Mills, Calif.
B. S., D. V. M., State College of Washington, 1935
Vouchers: Horace B. F. Jervis and Wm. R. Kermen.

CLARK, CHESTER F. 508 Charles St., East Lansing, Mich.
D. V. M., Michigan State College, 1929
Vouchers: B. J. Killham and E. T. Hallman.

ERICKSON, IRWIN 7022 Brooklyn Ave., Seattle, Wash.
B. V. Sc., Ontario Veterinary College, 1929
Vouchers: S. W. Clark and A. J. Bonaci.

LIEBERMAN, LT. LEO L. Veterinary Station Hospital, Fort Ethan Allen, Vt.
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and J. H. Knapp.

Applications Pending

SECOND LISTING

(See November, 1935, JOURNAL)

Adams, Edward B., Menno, S. Dak.
Daman, Lt. Arthur H. L., 217 Roup Ave., Pittsburgh, Pa.
Enas, Jack R., 638 E. Water St., Lock Haven, Pa.
Farkas, Albert B., 4321 N. Crawford Ave., Chicago, Ill.
Kerr, Lt. George M., 8 Garfield Ave., Hyattsville, Md.
Koch, Bernard, 621 W. Lombard St., Baltimore, Md.
McCorkle, Harold C., Redmond, Wash.
Macdonald, Hugh E., Veterinary Research Station, Box 639, Lethbridge, Alta.
Michel, Herman A., Carthage, S. Dak.
Ruehle, Otto, Jr., 1710 S. E. Belmont St., Portland, Ore.
Ruggles, George A., 8821 Aurora Ave., Seattle, Wash.
Spayth, Guy V., Bloomville, Ohio.
Studer, Sebastian N., Steamboat Springs, Colo.
Wohnsiedler, George, 129 Church St., Carthage, N. Y.
Woodhouse, Clarence A., 914 Studer Ave., Columbus, Ohio.

The amount which should accompany an application filed this month is $5.42, which covers membership fee and dues to January 1, 1936, including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.42, the additional $5.00 being for the 1936 dues.

STATE BOARD EXAMINATION

Kansas State Board of Veterinary Examiners. State House, Topeka, Kans. January 14, 1936. Further information may be obtained from the Secretary. Dr. Thos. P. Crispell, Secretary, Parsons, Kans.

COMING VETERINARY MEETINGS

Horse and Mule Association of America. Palmer House, Chicago, Ill. December 4, 1935. Mr. Wayne Dinsmore, Secretary, 407 S. Dearborn St., Chicago, Ill.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. December 4, 1935. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. December 4, 1935. Dr. Milton R. Fisher, Secretary, 4405 W. Pine St., Saint Louis, Mo.

National Association of B. A. I. Veterinarians. Hotel La Salle, Chicago, Ill. December 4-6, 1935. Dr. F. A. Imler, Secretary, Box 187, Kansas City, Kan.

United States Live Stock Sanitary Association. Hotel La Salle, Chicago, Ill. December 4-6, 1935. Dr. O. E. Dyson, Secretary, Live Stock Exchange Bldg., Wichita, Kan.

East Tennessee Veterinary Medical Society. White Surgical Supply Building, Knoxville, Tenn. December 7, 1935. Dr. Robert L. Hummer, Secretary, 312 W. Church Ave., Knoxville, Tenn.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. December 10, 1935. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

San Diego County Veterinary Medical Association. San Diego, Calif. December 10, 1935. Dr. L. K. Knighton, Secretary, 3438 Mountain View Dr., San Diego, Calif.

Nebraska State Veterinary Medical Association. Evans Hotel, Columbus, Neb. December 10-11, 1935. Dr. E. C. Jones, Secretary, c/o Norden Laboratories, Grand Island, Neb.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. December 11, 1935. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Western New York Veterinary Medical Association. Buffalo, N. Y. December 12, 1935. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. December 17, 1935. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. December 18, 1935. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. December 18, 1935. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

Delaware Veterinary Medical Association. University of Delaware, Newark, Del. December 27, 1935. Dr. C. C. Palmer, Secretary, University of Delaware, Newark, Del.

American Association for the Advancement of Science. Saint Louis, Mo. December 30, 1935-January 4, 1936. Dr. Henry B. Ward, Secretary, Smithsonian Institution Bldg., Washington, D. C.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 6-7, 1936. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

Wisconsin Veterinary Medical Association. Madison, Wis. January 6-8, 1936. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 7-8, 1936. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 7-10, 1936. Dr. Cliff D. Carpenter, Secretary, 337 Central Ave., Los Angeles, Calif.

Maine Veterinary Medical Association. Waterville, Me. January 8, 1936. Dr. R. E. Libby, Secretary, Richmond, Me.

Minnesota State Veterinary Medical Society. Nicollet Hotel, Minneapolis, Minn. January 8-9, 1936. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 9-10, 1936. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 9-10, 1936. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Vermont Veterinary Medical Association. Montpelier Tavern, Montpelier, Vt. January 11, 1936. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. January 13, 1936. Dr. J. N. McIlnay, Secretary, 3251 Leavenworth St., Omaha, Neb.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 13-15, 1936. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Rhode Island Veterinary Medical Association. Narragansett Hotel, Providence, R. I. January 14, 1936. Dr. J. S. Barber, Secretary, 14 Washington St., Central Falls, R. I.

Kansas Veterinary Medical Association, Manhattan, Kan. January 14-15, 1936. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Arkansas Veterinary Medical Association. (Joint meeting with the Tennessee Veterinary Medical Association.) Memphis, Tenn. January 20-21, 1936. Dr. T. M. Dick, Secretary, City Hall, Little Rock, Ark.

Tennessee Veterinary Medical Association. Memphis, Tenn. January 20-21, 1936. Dr. A. C. Topmiller, Secretary, Department of Agriculture, Nashville, Tenn.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 21, 1936. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 21-23, 1936. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Iowa Veterinary Medical Association. Fort Des Moines Hotel, Des Moines, Iowa. January 21-23, 1936. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Colorado Veterinary Medical Association. Albany Hotel, Denver, Colo. January 23, 1936. Dr. B. R. McCrory, Secretary, Colorado State College, Fort Collins, Colo.

Mississippi State Veterinary Medical Association. Lamar Hotel, Meridian, Miss. January 23-24, 1936. Dr. E. H. Durr, Secretary, Clinton Blvd., Jackson, Miss.

Nevada State Veterinary Association. Reno, Nev. January 24, 1936. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 27-31, 1936. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 28-30, 1936. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

INFECTIOUS MYXOMATOSIS OF DOMESTIC RABBITS*

By F. D. McKENNEY *and* J. E. SHILLINGER

Bureau of Biological Survey, U. S. Department of Agriculture, Washington, D. C.

Infectious myxoma of rabbits was reported first by Sanarelli,[1] as occurring in South America. The disease, as determined by early workers, is caused by a filtrable virus, highly contagious and frequently fatal to the domestic rabbit. It was so named by Sanarelli, because of the tendency of the disease to produce myxomatous-like tumors in the skin. While known, since 1898, to occur sporadically in Mexico, the disease was not recorded in the United States until 1930, when Kessel and co-workers[2] reported it as occurring in commercial rabbitries of California. Since that time, the disease has made its appearance each summer in certain localized areas of southern California, and while it has occurred in only a small percentage of rabbitries in the state, its extreme infectiousness and uniform fatality have caused severe losses in those affected.

Although infectious myxomatosis has been diagnosed at times in most of the coastal counties of California south of San Francisco, there are two areas which seem to be regularly productive of the disease. The western parts of Santa Barbara and Ventura Counties constitute one of these, and the second is a small area confined to the southwestern fourth of San Diego County. The most inland point of occurrence at any time has been the city of Bakersfield, and the epizoötic there was found to be correlated with an importation of rabbits from the vicinity of Santa Barbara. It is quite possible, however, that small epizoötic have occurred elsewhere in the United States, but were not recognized.

SYMPTOMS

The first objective symptoms of infectious myxomatosis are a white, purulent lacrimation and acute blepharitis. There is a pronounced edema of the ears, which in some cases causes them to become pendant. The edema of the face, lips and nose makes the head massive and accounts for the lay term of "bighead" often applied to the disease. There is severe congestion and edema of all external body orifices, the fur is rough, and the eyes lusterless. Through experimental or spontaneous infection,

*Received for publication, July 18, 1935.

the disease is regularly fatal and less than 1 per cent of normal unprotected animals recover from this disease.

In less severe or non-fatal cases, the edema, which constitutes a prominent part of the picture, gradually recedes and leaves in its place, nodules of varying sizes (fig. 1). The nodules have a gross appearance of papillomas, their surface being rough and denuded of hair, but they are firmly fixed to the underlying tissues. The nodules most commonly occur on the ears or head, but may also be found on the legs or feet. During the initial stages of the disease, the body temperature is increased and, according

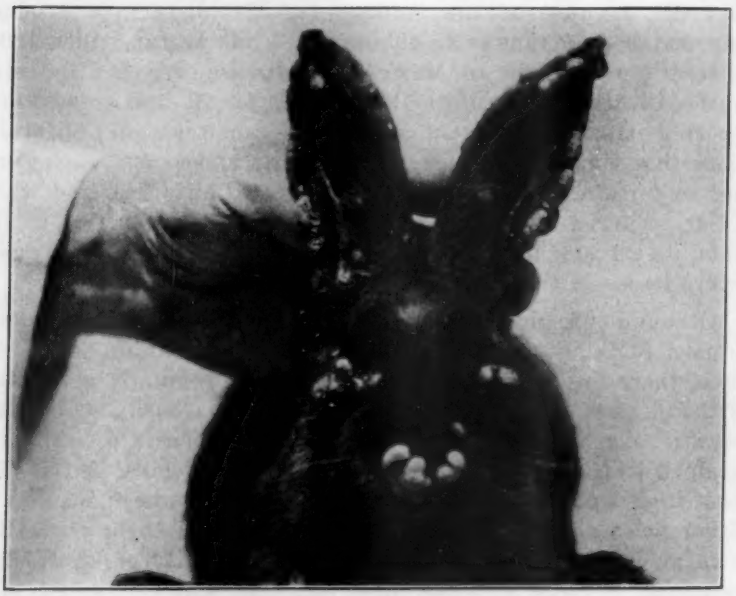

FIG. 1. Rabbit affected with myxomatosis showing edema and nodular excrescences.

to Hobbs,[3] there is a progressive increase in the white cells of the blood, but the differential count remains unchanged during the course of the disease.

The edema of the ears, face and body orifices increases as the disease progresses; affected areas are firm to the touch and will not pit under pressure.

In the terminal stages of the disease, the animal becomes comatose, breathing is labored, and there is a purulent discharge from the eyes and nostrils (fig. 2). Death ensues in two to four days after the first symptoms of the disease are manifest and is usually directly attributable to a secondary pneumonia.

Incubation Period

The incubation period of the disease is from eight to eleven days, symptoms most commonly appearing on the ninth or tenth day after inoculation. In this respect the strains of myxomatosis virus isolated in California seem to be somewhat less virulent than the South American strains of the virus, as other investigators working with the South American strains report an incubation period of three to five days.

The disease is easily transmitted through contact with affected animals or by placing animals free of the disease in hutches or

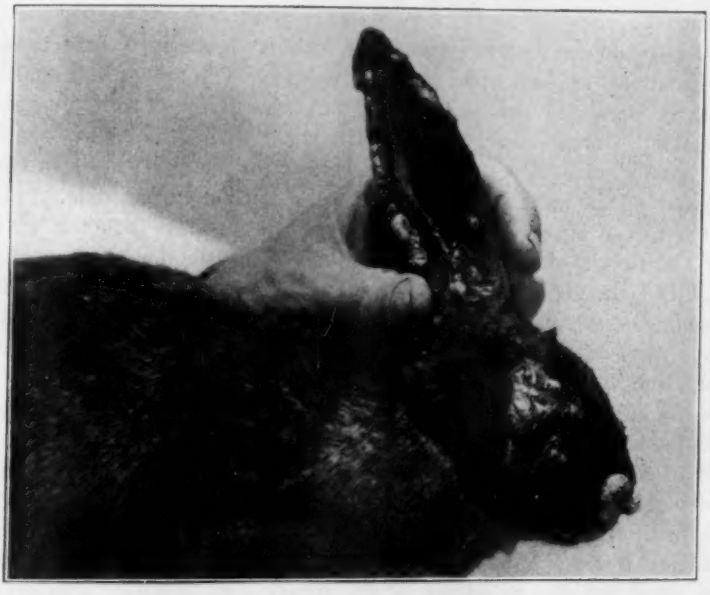

Fig. 2. Side view of animal in figure 1 showing purulent discharge from eyes and nose.

cages formerly occupied by infected animals. It can also be transmitted regularly by injecting, subcutaneously or intravenously, small quantities of blood, liver, spleen or lymph-node. Infection was accomplished also when small quantities of those infective tissues were rubbed on the gently scarified skin of a normal rabbit. The purulent discharge from the eyes and nose is infective if placed in the eye or brought into contact with surfaces covered by mucous membranes, *i. e.*, nose, vagina, mouth, or intestinal tract. The urine from infected animals is innocuous when injected subcutaneously, according to Hobbs.[3]

Although experiments have failed to detect any difference in the infectiousness of the virus of myxomatosis for various breeds of the domestic rabbit, it does not appear to be infective for other animals. All attempts to infect chickens, guinea pigs or pigeons with the virus were without positive results. Hobbs[4] reports that various species of wild rabbits also carry an immunity to the disease. He attempted to infect individuals of the black-tailed jack rabbit (*Lepus californicus* Gray), the varying hare (*Lepus americanus* Erxleben), and the common wild cottontail (*Sylvilagus transitionalis* Bangs), but each species proved to be immune.

PATHOLOGY

A postmortem examination of a rabbit dead with infectious myxomatosis reveals little gross pathology. The spleen is usually slightly enlarged and congested, but the liver is not grossly affected. Hobbs[3] reports that with the South American strain of virus, macroscopic white flecks appear on the surface or in the interior of the spleen of rabbits affected with myxomatosis. This has not been observed so far in any of the animals infected with the California strain. The mesenteric lymph-nodes are usually slightly enlarged and may show slight hemorrhage. When sectioned, the edematous areas present a glistening, white, gelatinous appearance, which areas bulge slightly on the cut surface and from which a clear serous fluid can be expressed.

Animals in which the disease has progressed to the later stages usually show some type of pulmonary pathology. This may consist of a hypostatic congestion, broncho-pneumonia, or complete consolidation of the entire lung. Since these changes are lacking in those animals that are sacrificed in the early stages of the disease, however, it is felt that these pathological changes in the lungs are due to edema of the upper air-passages, a general lowering of vitality, and are not produced by the virus of myxomatosis.

A study of the histopathology of infectious myxomatosis reveals that the pathological changes confined largely to skin and adjacent tissues show large areas of mucin-like material, through which is found a loose stroma of fine fibrils. Large stellate cells are found throughout the area, the nuclei of which are large and granular, and contain multiple nucleoli. It is often difficult to distinguish the contour of the cytoplasm of these myxoma-like cells from the background of homogenous staining material.

Other areas are found in which the reaction seems to be more inflammatory than neoplastic; in such areas the cellular elements

are made up of polymorphonuclearleukocytes or fragments of these cells and collections of lymphocytes and monocytes within a network of hyperplastic connective tissue. The epithelial cells of the skin show some destruction, but the picture is one of both hypertrophy and hyperplasia, making this layer of cells many times thicker than in a normal section of skin. Rivers[5] and others working with the South American strain of the myxoma virus have placed much emphasis on the occurrence of intracellular masses or inclusion bodies in the epidermal cells. This has not been a consistent or prominent part of the histopathology in animals inoculated with the California strain.

IMMUNOLOGY

In an attempt to protect rabbits against the fatal myxoma virus, some interesting problems have presented themselves. Fisk[7] and others, in attempts to immunize animals by means of chemically inactivated virus, reported little success with such methods, while Hobbs[3] found that there were no demonstrable virucidal or protective properties in the heated blood from infected rabbits. Shope,[6] however, demonstrated, in his work with a tumor-producing virus isolated from the cottontail, that when this tumor was transplanted into domestic rabbits, it underwent absorption in the animals after a time and that such animals were usually immune to inoculation with infectious myxomatosis. He further reported that while serum from animals infected with the filtrable-virus tumor carried no protection against infectious myxomatosis, the animals that had recovered from infectious myxomatosis, by means of previous protection with injections of the filtrable-virus tumor, did show virucidal properties in their sera. Of a series of 15 animals that he had protected previously by the filtrable-virus tumor and later inoculated with the virus of myxomatosis, only one died of the disease, two others showed complete resistance, while the remainder showed clinical evidence of infectious myxomatosis but later recovered.

In an effort to develop a practical method whereby large numbers of domestic rabbits could be immunized against infectious myxomatosis, a sample of the filtrable-virus tumor was obtained and a series of ten animals were inoculated. The inoculum was prepared by finely mincing a piece of glycerin-preserved tumor with scissors, then grinding with sterile sand and sufficient physiological saline to make approximately a 5 per cent solution. The material was allowed to stand until the coarser particles had settled to the bottom. The supernatant fluid then was de-

canted off and used as a source of virus. The animals received injections of 1 cc intratesticularly and 2 cc subcutaneously. Contrary to results obtained by Shope, five of these animals failed to show any evidence of infection other than a transitory swelling at the site of the injection. The remaining five animals, within five to seven days, showed varying degrees of tumor growth, but in each case the growth was more rapid in the testicle than in the subcutaneous tissue.

Fifteen days after being inoculated with the tumor virus, the entire group of ten animals were inoculated with the virus of myxomatosis and as controls four normal animals also were inoculated. For this inoculation 1 cc of a 1:50 dilution of the myxoma virus was injected subcutaneously. The four controls and the five animals that failed to show a growth of the filtrable-virus tumor died with typical symptoms of myxomatosis in eight to eleven days. One animal that showed only a small testicular tumor, which contained hard shot-like nodules, also died as a result of the injection of myxomatosis virus, although the fatal termination of the disease was delayed in this case until 15 days. Occasionally normal animals injected with the myxoma virus live until the 14th or 15th day, so that it is not known that the delay in the fatal termination of the disease was a result of infection with the filtrable tumor. Two of the remaining cases were completely resistant to the inoculation with the virus, while the other two showed clinical evidence of the disease but recovered.

An attempt then was made to immunize domestic rabbits to infectious myxomatosis by use of a desiccated tumor virus. The animals were inoculated by injecting 1 cc of the desiccated tumor virus suspended in physiological saline intratesticularly. Five animals were inoculated with the virus, but none showed gross evidence of being infected with the filtrable tumor. These animals then were injected with myxoma virus and all died with acute symptoms of the disease.

Considerable difficulty was experienced in transmitting the tumor virus in series, although tumors of various ages were used for transferring the virus; after one or two serial transfers of the tumor virus through domestic rabbits, it could not be recovered or was attenuated sufficiently that it would no longer produce gross tumors. An experiment then was conducted to determine whether the attenuated tumor virus would protect animals against the virus of myxomatosis when there was no gross evidence of tumor growth. Seven animals, checked by two

controls, were inoculated with the virus of a tumor that had been passed through one series of domestic rabbits. The selected tumor had given the most consistent results as to growth and immunizing power of any of the strains of virus used. The tumor had been implanted in the testicle of a rabbit for 27 days and showed evident signs of decrease in size.

The virus was prepared in the usual manner by grinding with sterile sand and physiological saline. Two cc of a 10 per cent solution of the virus was inoculated intratesticularly and subcutaneously into seven animals. Only one of the seven showed gross evidence of being infected with the tumor virus. On the tenth day after inoculation with the tumor virus, each of the seven animals received 1 cc of a 1:100 dilution of myxomatosis virus. The animal in which there was gross evidence of tumor infection proved to be immune; however, one animal in which there was no evidence of tumor infection also proved to be resistant to the virus of myxomatosis and showed no evidence of the disease. The five remaining animals and the two controls died with myxomatosis, showing typical symptoms of the disease. In one animal, the fatal termination of the disease was delayed until the 20th day.

Although no definite conclusions can be drawn from the use of such a limited number of animals, in view of the almost uniform fatality of the disease, this result might be interpreted as an indication that the tumor virus could be incapable of producing a palpable tumor in the testicle of a rabbit yet have sufficient virulence to give some protection against the virus of myxomatosis. It is more probable, however, that the animal possessed a natural immunity to both the virus of infectious myxoma and the filtrable-tumor virus.

The consistency with which the tumor virus protects against inoculation with myxomatosis, however, seems to be in direct proportion to its ability to produce actively growing tumors. This is confirmed to some extent by the fact that animals that have been infected with the filtrable-tumor virus and show gross evidence of intratesticular tumors may lose their resistance to an infection with the virus of myxomatosis after retrogression in the tumor takes place, and the viable tumor virus can no longer be demonstrated.

Groups of animals that had been infected with the tumor virus for varying lengths of time rarely showed resistance to the virus of myxomatosis after the tumor had undergone absorption. Shope has shown that animals once infected with the tumor virus are

immune to further infections of the same virus, but this immunity, however, evidently does not protect them against infectious myxomatosis.

Since Fisk and Kessel,[7] as also reported by Kessel, Fisk and Prouty,[8] had failed to demonstrate protective properties in the attenuated virus of myxomatosis or virucidal properties in heated or chemically treated blood or serum of rabbits with the disease,* an attempt was made to demonstrate these properties in tissues, blood or serum of animals that were immune or had recovered from the disease.

Normal animals were infected with the virus of myxomatosis, and after two days the animals received 5 cc of serum from animals immunized to the disease and which had received inoculations of virus many times larger than the minimum lethal dose. Since this procedure gave no protection against the disease, another group of animals were tried in which the serum was administered on the same day the virus was injected; still others received the serum from immune animals prior to the injection of virus. The amounts of virus and serum were varied, giving as much as 9 cc of serum in attempts to protect rabbits that had received 1 cc of the virus. Tissue extracts from immune animals then were tried in this manner for protective qualities, but all of these experiments were uniformly negative.

As Hobbs[4] had reported that it was possible to attenuate the virus of myxomatosis with serum from animals that were immune to the disease, it was decided to test the resistance of animals to myxomatosis virus after they had received injections of virus attenuated in such a manner. A 1:50 suspension of virus was prepared in the usual manner, and serum was obtained by drawing a quantity of blood from immune animals and allowing it to coagulate. Equal amounts of the clear serum and of the myxomatosis virus were placed in test tubes and allowed to stand at room temperature for three hours, and were then placed in the ice-box over night. Four animals were inoculated subcutaneously with 1.5 cc of the serum-virus mixture, but all four failed to show any symptoms of the disease within the usual time. Three weeks subsequent to the first injection, each of the four animals received an injection subcutaneously of 1 cc of the same virus that had not been treated with the serum. All four of the animals died with typical symptoms of myxomatosis.

*This work, as reported at the Fifth Pacific Science Congress, was financed by the Bureau of Biological Survey while C. C. Prouty and Roy T. Fisk were employés of that organization.

CONTROL

Severe losses from infectious myxomatosis in a rabbitry make drastic measures very necessary in order to control the disease. With our present knowledge of the disease, it is not advisable to attempt curative measures, but all animals showing symptoms of the disease should be destroyed immediately and the carcasses burned or buried under several feet of earth. Hutches, feeding utensils, and water crocks used by infected animals should be thoroughly disinfected. The thermal death point as reported by Hobbs[3] is about 50° C., so it is relatively sensitive to heat. The virus is known to be destroyed by 1.0 or 2.0 per cent solutions of carbolic acid after four days, so, where disinfectant solutions are used, they should be employed in sufficient concentration to be effective immediately.

COMMENT

Confirming the data of earlier workers, the writers succeeded in regularly transmitting infectious myxomatosis by using the exudate from the eyes or nose of infected animals and by small amounts of blood and tissue from animals with the disease. The disease is probably disseminated in this exudate and this probably accounts for the rapid spread of the disease after it has once been introduced into a rabbitry. The periodicity of the disease, however, might indicate that certain animals, although showing no clinical evidence of the disease, could become carriers of the infection or that there are other unsuspected reservoirs from which insect or other vectors could transmit the disease during the warmer months of each year.

Shope's success in immunizing domestic rabbits against infectious myxomatosis, by previously infecting the animals with the tumor virus, could not regularly be duplicated. While it was found that rabbits that were carrying an actively growing filtrable tumor or a filtrable tumor in the early stages of retrogression were often immune to the virus of myxomatosis, those rabbits in which the filtrable tumor had completely regressed were only infrequently found to carry any immunity to the virus of myxomatosis. It was further found that regardless of frequent animal passage in domestic rabbits, the filtrable-tumor virus rapidly decreased in virulence.

The ability of serum from rabbits immune to infectious myxomatosis to neutralize the virus of myxomatosis and yet fail to show curative or protective qualities gives rise to interesting speculations; however, there has not been a sufficient amount

of experimental work performed with the immune serum to draw definite conclusions. Myxomatosis virus neutralized by chemicals, heat, or immune serum usually shows no power of inducing immunity in normal rabbits, as when the attenuated virus fails to produce the disease, it does not affect the susceptibility of the animal.

SUMMARY

Infectious myxomatosis, a contagious fatal disease of domestic rabbits, has been described. Shope's filtrable tumor of wild rabbits as a practical means of immunizing animals against infectious myxomatosis has not as yet proved successful because of the difficulty in maintaining the filtrable-virus tumor in domestic rabbits and because they are not uniformly susceptible to the virus of the filtrable tumor. Domestic rabbits with gross filtrable-virus tumors are usually immune to the virus of myxomatosis. It is possible to attenuate or destroy the virus of myxomatosis with serum from rabbits that are immune to the disease, but the inoculation of virus so treated will not produce demonstrable immunity. Serum from immune rabbits has, so far, failed to induce protective qualities in the serum of normal rabbits against the virus of myxomatosis.

REFERENCES

[1]Sanarelli, G.: Das Myxomatogene Virus. Cent. Bakt., Abt. I, xxx (1898), p. 865.

[2]Kessel, J. F., Prouty, C. C., and Meyer, J. W.: Occurrence of infectious myxomatosis in southern California. Proc. Soc. Exp. Biol. & Med., xxviii (1931), p. 413.

[3]Hobbs, J. R.: Studies on the nature of infectious myxoma virus of rabbits. Amer. Jour. Hyg., viii (1928), p. 800.

[4]Hobbs, J. R.: The occurrence of natural and acquired immunity to infectious myxomatosis of rabbits. Sci., lxxiii (1931), p. 94.

[5]Rivers, T. M.: Changes observed in epidermal cells covering myxomatous masses induced by virus myxomatosum. Proc. Soc. Exp. Biol. & Med., xxiv (1927), p. 435.

[6]Shope, R. E.: A filtrable virus causing a tumor-like condition in rabbits and its relationship to virus myxomatosum. Jour. Exp. Med., lvi (1932), p. 803.

[7]Fisk, R. T., and Kessel, J. F.: Immunization studies with the virus of infectious myxomatosis. Proc. Soc. Exp. Biol. & Med., xxix (1931), p. 9.

[8]Kessel, J. F., Fisk, R. T., and Prouty, C. C.: Studies with the California strain of the virus of infectious myxomatosis. Proc. 5th Pacific Science Congress, pp. 2928-2939.

BUREAU TRANSFERS

DR. GORDON C. KENDALL (A. P. I. '28), from Mayaguez, Puerto Rico, to Chicago, Ill., on meat inspection.

DR. C. R. BEHLER (Chi. '04), from Kansas City, Mo., to Cincinnati, Ohio, on meat inspection.

DR. FRANK L. CHASTAIN (Ga. '30), from Oklahoma City, Okla., to Moultrie, Ga., on meat inspection.

DR. B. J. STOCKLER (O. S. U. '08), from South Saint Joseph, Mo., to Buffalo, N. Y., on meat inspection.

DR. D. S. KAY (San Fran. '11), from South Saint Paul, Minn., to Seattle, Wash., on meat inspection.

STERILITY IN COWS*

By C. H. CASE, *Akron, Ohio*

Sterility in dairy cattle will always be a big field for study because of the forced production methods that are used by our various breeders inhibiting the reproduction of offspring. If you look at the records of the different breed associations, you will find that the forcing of cows to give large milk production results in a great many never producing a living calf.

I have a client who paid $5,100 for a cow. She had one living calf at the time she was bought by this client and she had one more after she came to the farm. In the best day of a seven-day record she milked 149.2 pounds of milk. That is almost 18 gallons a day. That cow has never had a living calf since, although it has been about five years since the record was made. Forced production has a marked effect on sterility and makes us wonder what should be done.

Another thing that started me to work on this question of sterility was a banker who owned a farm that produced milk for a hospital, so that it was necessary to have the same amount all the year 'round. This banker came to me about 25 years ago and said, "Can you tell me some way by which I can maintain the breeding record every month and also have the same amount of milk each month?"

With that idea in mind, I started work with his herd and have gradually worked out a plan to get as near as possible a perfect herd for 38 of my clients. I have tried to devise some means of curing sterility, or at least, alleviating the condition.

I keep complete records. Every cow in the herd is listed on a separate page. Her breeding record is given. A notation is made every time she is bred. Two months later, she is examined for pregnancy, and that is noted. A notation is made when she freshens; when she expels the placenta, and if she does not come in estrum about two months after freshening, an investigation is made to learn why. We do not let it go for four or five months.

We make it a practice to visit these dairies once a month. You will find that they will be glad to pay you a certain sum to come to the farm every month and correct these conditions before it is too late, or before certain changes have taken place.

Dr. Williams tells us that to have a perfect breeding record a cow should have a calf every twelve months. I do not know

*Extemporaneous remarks delivered before the Section on General Practice, at the seventy-second annual meeting of the American Veterinary Medical Association, Oklahoma City, Okla., August 27-30, 1935, in the absence of the speaker who was scheduled to present this subject.

whether any of you keep records of your herds, but if you do, you will be surprised to find how few herds maintain a perfect breeding record. I have had only two herds in all my experience that had such a record.

If my records show that a cow has been served three times, the owner is instructed to hold her until my next visit, so that I can examine her and see what is wrong. She may have a cystic ovary, or something else may be the matter. If, on examination, it is found that a corpus luteum has been retained in one ovary, when you remove that and wash out the uterus, you have done everything I know of for the cow at that time, and the chances are that she will conceive.

When I have removed the corpus luteum, I inform the owner that the cow should be in estrum in three to ten days.

I have kept records on some 300 cows in different herds. One-third of the cows from which the corpus luteum had been removed conceived in five days, another third in 25 days, and the other third, with the third breeding, or 21 days later. It is fairly safe to say that one-third of the cows will breed at the first period after the removal of the corpus luteum.

When you remove a corpus luteum and do not douche the uterus, you have not done everything you should for the cow. When you catch the return flow in the hand, you will be surprised to find how many times there will be flakes of pus that you would never see any other way. I think one reason why a great deal of the sterility work fails is because the uterus is not washed out and the pus removed, no matter how mild the amount.

If you have the history of the cow and know she has been bred several times, and if you do not remove the corpus luteum, there is little chance of the cow becoming pregnant. The method I use for removing them is as follows: With one hand in the vagina and one in the rectum, using moderate force, I try to get a retained corpus luteum between my thumb and finger and expel it. After you have done that, you have done everything possible to induce pregnancy. It is a difficult process, particularly with heifers, but the thing to do is to return at the time of estrum. You must remove the corpus luteum or you will not get the desired results.

Another thing that causes sterility in heifers is vaginitis. You will be surprised at the condition you often will find in heifers that will not breed. By douching with a chlorine solution at regular intervals to remove the mucus, and particularly a day or so before estrum, you will find that a great many of those

heifers will breed right away, or at least the greatest percentage of them will.

If, after the removal of the corpus luteum, the owner does not notice the cow to be in estrum even in three to 21 days, and upon examination, another corpus luteum is found and it is expelled easily, the owner is instructed to breed the cow on the fifth day whether she shows any estrual period or not. A great many of them will conceive under such conditions. I can cite you a great number of instances wherein cows have conceived that did not show any estrual period at all. Some cows do not show estrum.

In sterility work, you never should put your hand in the vagina if you can possibly avoid it. You can do more harm by the infection you may carry into the vagina than all the good you can do by douching. Use an instrument of some kind, a glass tube, or the Carlson speculum. Any man who is going to do this work should have a Carlson speculum so as to be able to examine the cervix. In that way you have a light and can see if there is cervicitis or inflammation of the vagina. You can pull the cervix back and you never have to put your hand in the vagina. In that way you do not carry infection into the vagina, and your results will be greatly increased.

Years ago, before any different method was known, I used long forceps and the hand. Many a time I have had an owner tell me that after that method had been used, the cow discharged a lot of pus. Vaginitis had set in as the result of using my hand. The less of that one does, the better.

In cases that show a little pus in the return flow from the uterus, I use a 1:1,000 metaphen solution with a long-nosed 12-cc hypodermic syringe. I leave it within the uterus, and my results have been a great deal better since I have done that with such cases. If there is a considerable amount of pus, I use about 25 cc of concentrated chlorine solution. An injection of that will often clear up a condition that one had not been able to cope with.

I used to think there was nothing like Sterilac antiseptic for douching. You have heard me talk about that. I thought it kept its strength. I have spent hundreds of dollars for Sterilac. A few years ago, our county doctor told me what he was using, H. T. H., and since then, that is what I have used. The formula is on the outside of the can. It doesn't cost much—$1.48 a can, I believe, and a can makes ten gallons of solution. That is di-

luted, one ounce of the solution to a gallon of water. It is possible to make it even stronger than that without doing any harm. It makes the best solution I have ever used. It is mixed with soda, which makes a non-irritating solution.

When there is a great deal of pus in the uterus and you want to prevent sterility, a rubber catheter is the only safe thing to use. Insert the rubber catheter into the uterus, pull the uterus back, and with one hand in the rectum, put in a little water and then siphon it back out. Such cases clear up in a short time under that treatment. The next month, when you wash them, they will not show any discharge, and the gain in milk production is surprising.

One cow that I treated that way was giving 20 pounds of milk a day. She was sick, but within ten days she was giving 60 pounds and within 20 days, 80 pounds.

This use of the rubber catheter will do a great deal of good by removing the infection from the uterus and increasing milk production.

In treating sterility, the bull must be considered, too. The bull should be examined. It is very easy to do that, either by putting the hand in the rectum and pushing the semen out, or if there is a cow that happens to be in estrum that day, you can get it from the vagina of the cow. Insert a catheter into the vagina and bring out the specimen and smear it on a microscope slide. It is no trouble at all to get the sperms in that way. If there is considerable motility, you do not have to worry much, but I always take the slide home and stain it according to Dr. Williams' method, and if a great many of these sperms have the tails off, I recommend that the owner stop using that bull.

That is one way of checking on the bulls. If you find quite a number of tailless sperms, it would be advisable to recommend that the bull not be used any more.

I do not know that I have anything more to say on the subject of sterility. The method I have outlined is the one we are using every day to get results. One thing I want to emphasize, however, is the importance of keeping records. Make a notation of the results with every cow. The owner will tell you that this or that is the case, or that such and such a thing happened. Very often I find, when an owner tells me about certain things, that it is another cow to which he has reference, and I am able to prove it by my records. If you will keep records, you will be gratified at the results.

If any of you have dairy herds, I would suggest that you get the owners to let you take them on a monthly basis. You can do the work a great deal more cheaply if you make a visit to the herd every month. In that way you can do many more things for them in the way of sterility work, blood-testing, testing for mastitis, and so forth. If it had not been for mastitis testing in the last two years, we would not be doing nearly the business we are doing today.

As I said two years ago, the loss from mastitis in cattle is equal to the combined loss from Bang's disease and tuberculosis. Recently there has been a report issued on cows that have gone to slaughter on account of the loss of a quarter, and the report verifies my statement.

Blood-testing for Bang's disease, mastitis testing, and sterility work dovetail, and I wish you would start that procedure for the dairymen. By so doing, you can increase the milk production and make it possible for the owner to make money. If you want to help a client who is a dairyman, you can do nothing better than to get his cows to conceive regularly and maintain a perfect breeding record for the herd.

One thing I did not say much about is cystic ovaries. Sometimes an owner will hold a cow over several estrual periods because he wants her to make a big milk record and for that reason does not want to breed her. That is usually when cystic ovaries develop. Sometimes at the first estrual period ova will form and will not break. When they become old, after months or years, they are hard to break, even with the hands in the vagina and rectum. It is possible to break them when you make monthly visits, early before the wall of the ovary becomes so thick it is impossible to break down the cyst.

I was afraid of the knife method Dr. Williams used. Dr. Frick suggested using a regular two-inch hypodermic bleeding needle. Now I put my hand into the vagina and through the rectum, get the cystic ovary, puncture the cyst with the needle and you can feel the fluid flow out into the vagina. When there are two or three different cysts, it is possible to get all of them, and in a great many cases the cows will conceive after the removal of those cysts and douching the uterus. Seventy-five per cent of cows with cystic ovaries have more or less pus in the uterus.

Discussion

Dr. M. M. Leonard: There is one point that I am sure Dr. Case would like to have made clear. There are two solutions, H.T.H. and H.T.H. 15. There might be some confusion in the minds of those present as

to the chlorine product. I think Dr. Case mentioned H.T.H. H.T.H. 15 is also available.

DR. CASE: I use H.T.H. and make it up according to the formula on the can, with the soda ash. We always use the same solution. All bacteria do not come from the udder. They come from other sources, pails and cans and cooling systems, and so on. We sell a great many gallons of that solution to the dairymen, made with one ounce to three gallons of water, for disinfecting milking utensils and so forth. One supply can be used to wash pails and cans, the aerator, and to wipe off the udder. It can be made very cheaply and the use of it helps the dairymen to produce a better quality of milk.

DR. J. D. JONES: Will you explain your method of treating retained placenta?

DR. CASE: All of our regular clients know that we have an unvarying rule that we never attempt to remove a placenta under 72 hours. In removing placentas on the full-time carrying of the fetus for nine months and ten days, one rarely has much trouble. Just a few of the cotyledons adhere. Those are the cases in which we get the best results. We do not use rubber gloves, but we use our hands and work off the placenta as carefully as possible. After the placenta has been removed, we take the same chlorine solution and with a stomach-pump and hose, we pump in the fluid, with the hand as near the base of the uterus as possible. We put in a quart or so, siphon it out, and continue that until a clear solution comes back.

If it is a case of seven or eight months of pregnancy, which is the worst possible case, very often it is not possible to remove the placenta in 72 hours. If you try it and there is a hemorrhage, you had better stop, because you are doing more harm than good and it will start an infection by opening a fresh wound.

We fill two or three capsules with this concentrated chlorine solution and put them in the uterus. We go back, probably on the second day, and usually we find that the cotyledon attachments have loosened and we can wash them out. Sometimes a contraction of the cervix will cause trouble, but usually it is possible to remove them on the fifth day.

We always instruct the owner that if the cow goes off feed, does not give much milk, and walks around stiff and sore, he should call us. As a rule, that will not happen until four to five days afterwards. We go back and insert a regular horse catheter into the uterus, empty both horns, put in a little solution, and repeat that process until all the fluid is out. Usually you will find a real red, fetid pus.

Cows in that condition are the ones that are so sick. You will be surprised to see how quickly they will come back and do wonderfully for their owner. If you don't remove the fluid, it is almost certain death for the cow. We sometimes use two or three gallons to accomplish this douching.

Oklahoma Membership in the A. V. M. A.

Year	*Members*	*Year*	*Members*
1923	56	1929	39
1924	52	1930	47
1925	39	1931	48
1926	36	1932	53
1927	34	1933	53
1928	39	1934	59
1935	107		

STAPHYLOCOCCI ASSOCIATED WITH MASTITIS*

By RALPH B. LITTLE *and* EDWARD J. FOLEY
Department of Animal and Plant Pathology
The Rockefeller Institute for Medical Research
Princeton, N. J.

Mastitis associated with staphylococci, although less common than streptococcic mastitis, may occasionally become very acute. In most herds the infection is confined to a single animal, generally a heifer or cow recently introduced or an individual shortly after parturition. The affected animal may completely recover, or a partial or serious injury to the diseased quarter may remain, or death may occur. Occasionally the udder becomes gangrenous, resulting in the sloughing of the diseased parts.

Guillebeau[1] found that staphylococci were responsible for severe udder inflammations and that some types liquefied gelatin. Savage[2] studied five cases of staphylococci mastitis. From his material he obtained 22 strains, of which 16 attacked mannite and 19 liquefied gelatin. Evans[3] reported that, in milk drawn from the udder, 58.8 per cent of the samples contained micrococci. Both pathogenic and non-virulent varieties were present in the skin, which were of the same types as those found in milk. The hemolytic aureus types appeared to be more pathogenic than the non-hemolytic albus strains. Jones[4] considered that next to streptococci, the micrococci were more frequently involved in udder disease than any other microörganisms. He intimated that prognosis in mastitis was more favorable with a staphylococcic infection than one caused by streptococci. The fermentative characteristics of 28 strains were given. Of these, 23 liquefied gelatin at 22° C. and 21 attacked glucose, lactose, saccharose and mannite. In three cows a chromogenic micrococcus was obtained from abscesses of the udder. This condition usually occurred in cows recently introduced into the herd. Carpenter[5] inoculated 2 cc of a 24-hour broth culture of non-hemolytic *Staphylococcus aureus*, obtained from a case of mastitis, into the udders of three heifers and two cows. Four animals reacted severely, showing a high temperature with loss of appetite for at least 36 hours following the inoculation. Ten days later, one cow died from septicemia resulting from the infection of the udder and at autopsy staphylococci were isolated from all the tissues of the body. In three animals abscesses appeared in the udders. Minett *et al.*[6]

*Presented at the seventy-second annual meeting of the American Veterinary Medical Association, Oklahoma City, Okla., August 27-30, 1935.

reported six cases of mastitis attributed to staphylococci and suggested that this type of mastitis was more severe than the usual form caused by streptococci, since two out of six cases died. In the majority of these, the infection occurred shortly after parturition. The microörganism on agar, solid serum or potato produced colonies which were dirty white or pale fawn in color and coagulated litmus milk with production of acid. Gelatin was liquefied after incubation, for two or three weeks at 15 to 18° C. Acid was produced in lactose, glucose, saccharose and mannite. Broth cultures were usually fatal to mice. Plasteridge *et al.*[7] found that in about 10 per cent of their cases of udder inflammation staphylococci were responsible. From the results of their observations it would appear that mastitis staphylococci attack mannite while the more harmless varieties do not.

During the past 17 years, the late Dr. F. S. Jones and one of us (R. B. L.) have occasionally encountered severe cases of staphylococcic mastitis in dairy cows and a certain number seem worthy of special mention. In 1918, Jones reported in some detail the clinical and bacteriological findings of a case of mastitis attributed to staphylococci. Since then a culture from this cow (C 60) has been carried in stock and is included in this publication so the case will be presented again. At the onset of mastitis the left hind quarter was firm, swollen and tender to manipulation. The secretion was thick, yellowish white, and contained 2,172,000 staphylococci per cc. The acute attack persisted for about ten days before the infection subsided. In 1932, we had occasion to examine a herd of 130 cows in which two animals had previously died as a result of mastitis. In six cows showing clinical symptoms of mastitis, staphylococci were the causative pathogens. Before the outbreak subsided four cows died and three others were sold later as unfit for milk production. During the course of the disease, the affected cows were very sick, depressed and had elevated temperatures. There was inappetence, with diminution in milk. The diseased quarters were swollen and firm, with a scanty blood-like secretion. In two cows the quarters became abscessed. Cultures of staphylococci designated as "Gray Cow 52" and "Gray Cow LF I" were obtained from individual quarters of two cows. On November 20, 1934, mastitis was observed in the right fore quarter of cow U 234. The quarter was swollen and firm with nearly a complete cessation of secretion. Two days later, mastitis was detected in the right hind quarter. The temperature was elevated and the cow appeared ill. Before she was destroyed, both quarters were very firm and the skin

had a bluish-red tinge. The mastitis was attributed to staphylococci and culture U 234 was recovered from the bloody exudate of the right fore quarter. Six days later, another cow in the same herd developed mastitis in the left fore quarter and died within 48 hours. Three samples of milk examined at 12-hour intervals during the course of the disease indicated that staphylococci were responsible for the infection and culture S 313 was isolated.

Since the small staphylococci are occasionally so pathogenic for cattle, it is difficult to understand why they are found in the milk of some individuals in enormous numbers without inducing death or serious inflammation of the udder.

In the daily examination of milk from three young cows over a period of six weeks, we have frequently encountered enormous numbers of typical staphylococci in the fore milk with very few organisms detectable in the remainder. The leukocyte count, the pH and the percentage of chlorides were also within normal limits. In a dilution of 1:200 of the fore milk in veal infusion blood-agar plates the count averaged between 10,000 and 60,000 microörganisms per cc with very few staphylococci present in the residual milk. Therefore this observation suggested that the growth of staphylococci under normal conditions was confined to the teat-canal, indicating that the milk in the secretory portions of the udder was not satisfactory for bacterial multiplication. It is assumed that the growth of the organisms in the udder was maintained at a low level by the action of some natural barrier present in the udder secretions.

In previous communications[8, 9] it has been shown that freshly drawn milk possesses a substance which is capable of inhibiting multiplication of bacteria for varying periods of time. It has also been proved that the substance varies in concentration in different cows and from quarter to quarter of the same animal. This is well illustrated in the following experiment. The milk from a cow with staphylococcic mastitis was examined bacteriologically with the result that the infection was found to be confined to the left hind quarter. The organisms were not present in the secretions from the three other quarters. When the milk from the left hind quarter was heated at 58° C. for 20 minutes (fig. 1) and then plated with 10 cc of veal infusion agar inoculated with approximately 1,000 staphylococci, no inhibition occurred. If, on the other hand, 3 cc of a mixed milk (fig 2) from the three normal quarters was plated in a like manner, complete inhibition occurred. If 1 cc of the milk from the left hind quar-

ter was mixed with 2 or 3 cc of the milk from the normal quarters (fig. 3), the organisms grew without hindrance. In another experiment, when 1 cc of normal cow serum was mixed with 3 cc of milk from the normal quarters (fig. 4), it was found that the milk lost its inhibitory action with the addition of the serum.

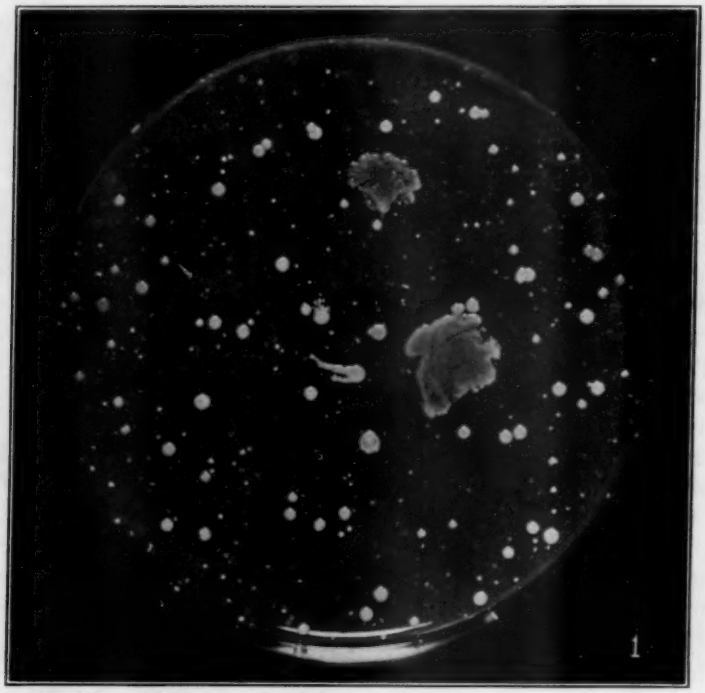

FIG. 1. Petri dish containing 3 cc of mastitis milk, heated at 58° C. for 20 minutes, and incubated for 24 hours with 10 cc of veal-infusion agar to which approximately 1,000 staphylococci had been added.

Recently a small staphylococcus has been recovered in pure culture from five superficial abscesses of the skin of the udder. An abscess associated with staphylococci appears as a small whitish or reddish vesicle on the outer epidermal layer of the skin. At the onset, it varies in size from a small millet seed to that of a pea. Once the abscess is opened or ruptured, it dries up and leaves a temporary, dry, scaly depression in the skin. On the other hand, the inflammatory process may become acute, involving deeper portions of the udder, causing considerable swelling of the quarter and pain at milking. The abscess is local, for the secretions are in no way altered during an attack. There

is no evidence at hand which suggests that staphylococci penetrate through the wall of the abscess and reach the secretory tissues. Since in one cow a pure culture of *B. pyogenes* was recovered from a deep udder abscess, it is evident that other organisms also may be responsible for the infection of the skin of the udder.

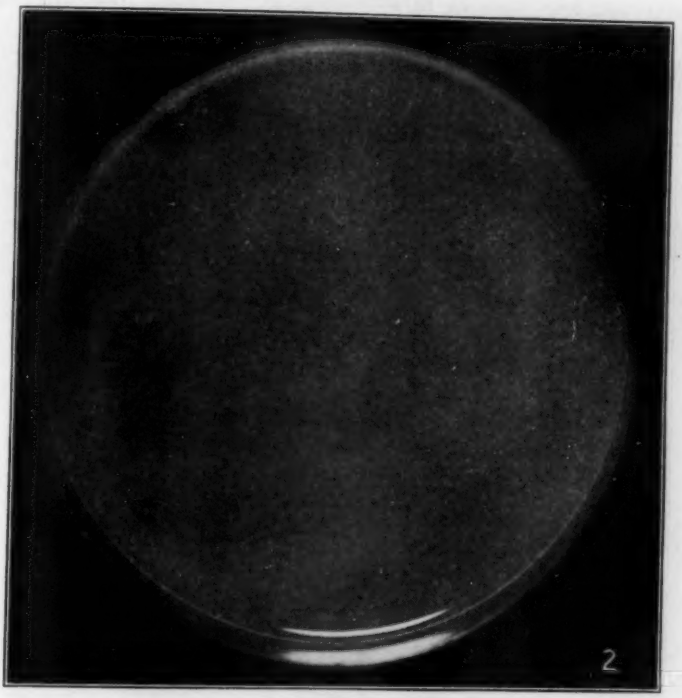

FIG. 2. Petri dish containing 3 cc of mixed milk from three normal quarters, heated at 58° C. for 20 minutes, and incubated for 24 hours with 10 cc of veal-infusion agar to which approximately 1,000 staphylococci had been added.

The typical colonies of staphylococci and those associated with abscesses of the skin are small and spherical in shape. On agar plates, after 24-hour incubation, the colonies are moist, raised, and of a grayish tint which usually changes to an orange or a light fawn color in a few days. The color characters are well defined on agar slants which have become rather dry or in milk-agar plates. Bouillon cultures, after 16 to 24 hours, are uniformly turbid, with the formation of a slight amount of soft sediment. In older cultures an adherent ring may appear around the tube at the upper surface of the broth. On fresh, sugar-free

blood-agar, surface colonies show, after 24-hour incubation, slight zones of beta hemolysis which on further incubation increase in diameter. Deep colonies as a rule show little if any hemolysis, except in heavily seeded plates. The microörganisms have a tendency to clump on films or form small chains of two to four segments. The individual staphylococci, since they measure on the average less than 1μ in diameter, are smaller than the non-

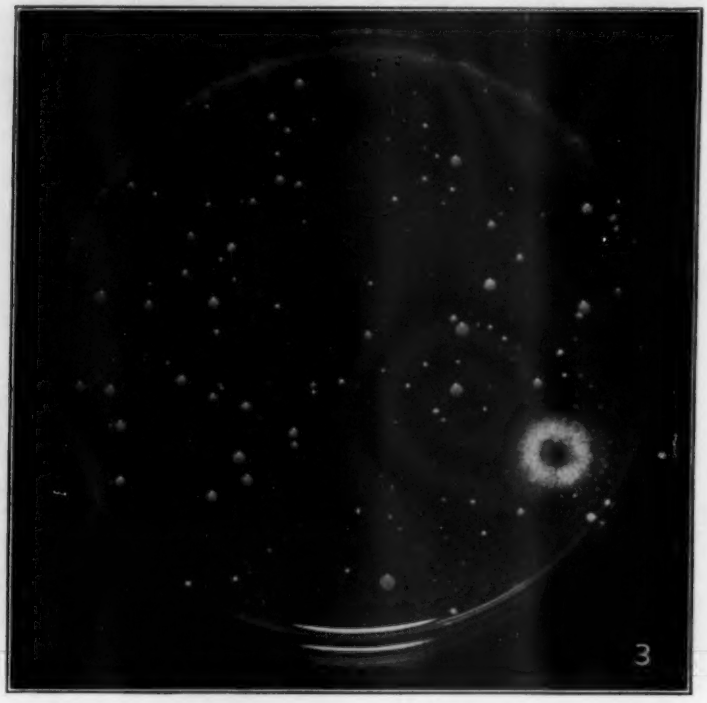

FIG. 3. Petri dish containing 1 cc of mastitis milk mixed with 2 or 3 cc of milk from normal quarters, heated at 58° C. for 20 minutes, and incubated for 24 hours with 10 cc of veal-infusion agar to which approximately 1,000 staphylococci had been added.

virulent strains. With the Gram stain the results are not constant and vary considerably with the age of the culture. If stained properly, most recently isolated strains give a Gram-positive reaction; whereas, with older cultures the reaction is less certain.

It was found that the cultures recovered from abscesses of the skin of the udder or from secretions of cows which died or had recovered from a staphylococcic mastitis were comparable in size

and pigment formation. It therefore seemed of interest to determine if the cultures had other characters in common. Hucker,[10] in his studies of the parasitic and saprophytic coccaceae, developed a classification and showed that certain tests proved to be of great assistance in separating the group into subgroups and species. These tests comprised: reactions in milk, presence or absence of chromogenesis, nitrate reduction, liquefaction of

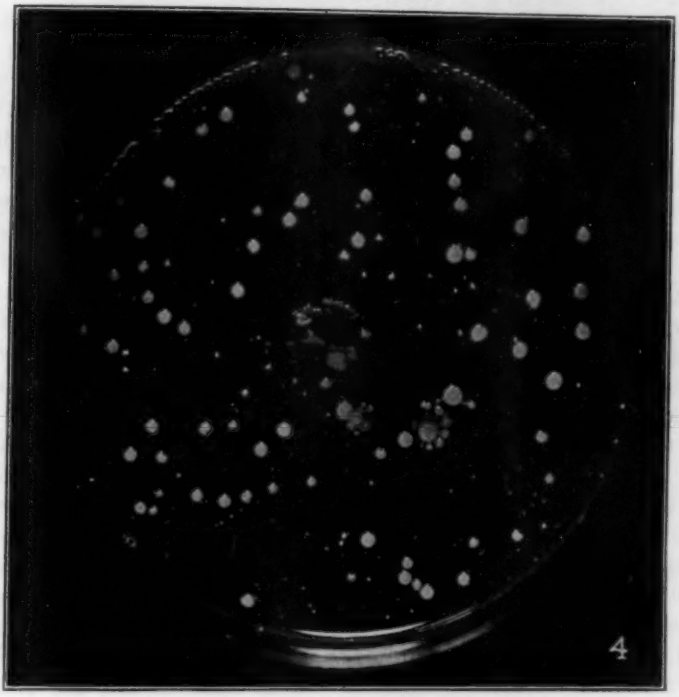

FIG. 4. Petri dish containing 1 cc of fresh, normal cow serum mixed with 3 cc of milk from normal quarters, heated at 58° C. for 20 minutes, and incubated for 24 hours with 10 cc of veal-infusion agar to which approximately 1,000 staphylococci had been added.

gelatin and the ability to utilize ammonium salts as the sole source of nitrogen. Breed[11] studied 177 cultures of micrococci isolated from udders and classified 171 of them, according to Hucker's system, into twelve groups. Thirty-three strains were regarded as being *Micrococcus aureus* and 21 as *M. albus*. These species were apparently established in the udders, since they were isolated over a period of some weeks. This same technic, in addition to certain other tests, was here applied to our organ-

isms. To obtain a complete description of the preparation and reactions of the reagents used by Hucker and Breed, reference to their papers may be made.

The cultural differences between the typical and a few miscellaneous strains are presented in table I.

In table I it is shown that twelve cultures of the small-type staphylococcus, isolated either from milk or udder abscesses, form a rather definite group. All strains are small in size, orange to a light fawn in color, and produce acid in dextrose, lactose, mannite and glycerin. Nitrates are reduced to nitrites and gelatin is liquefied. Of the twelve cultures studied, none were able to use ammonium phosphate as the sole source of nitrogen. Pathogenicity tests in mice demonstrated that only three strains failed to kill within 24 to 48 hours following intraperitoneal inoculations of 0.2 cc of young broth cultures. Of these, one culture was isolated by Jones in 1918 and the other two in 1932. With one culture, obtained in 1932, a mouse lived six days. It is possible that the two cultures (1 and 2) lost their infectivity by repeated transfers. It is recognized, according to Hucker's classification, that twelve out of 15 strains isolated from milk or associated with abscesses of the skin of the udder have the same cultural characteristics as *M. aureus*. Our cultures uniformly show the production of pigment on agar slants and milk plates. Cultures 13, 14 and 15 gave an atypical reaction in litmus milk and failed to kill mice. The cocci are larger than our typical strains and two produce hemolysis on blood-agar so they are not strictly comparable to the typical strains. With the remaining organisms (16, 17, 18, 19 and 20) the reactions differ in many respects. The pigment varies from light yellow or lemon yellow to orange. All attack dextrose and two fail to produce acid in mannite, while four reduce nitrates to nitrites. Three cultures can use ammonium phosphate as a source of nitrogen and, with the exception of culture 20, all fail to liquefy gelatin. In litmus milk three cultures react similarly to the typical strains; whereas, another fails to reduce litmus and one (18) produces a soft curd in milk. Four cultures are markedly hemolytic, while one shows no hemolysis. All strains fail to kill mice after 21 days. According to Hucker's classification, cultures 16 and 17 are designated as *M. varians*, 18 as *M. luteus*, 19 as *M. aurantiacus*, and 20 as *M. citreus*.

It is shown in the graph (fig. 5) that staphylococci, associated with abscess of the skin, when introduced into the udder, are capable of producing an acute severe infection presenting the

TABLE I—*The cultural reactions of 20 strains of pathogenic and saprophytic staphylococci.*

Culture	Source	Chromogenesis	Dextrose	Lactose	Mannite	Glycerin	Reduction of Nitrates to Nitrites	Liquefaction of Gelatin	Ammonium Phosphates	Hemolysis	Litmus Milk	Intraperitoneal Inoculation of Mice with 0.2 cc of Young Broth Culture
1	C 60 milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	—
2	Gray cow 52 milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	—
3	Gray cow I LF milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died 6 days
4	S 313 LF milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
5	U 234 RF milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
6	1932 LH abscess	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
7	1932 LH milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
8	1937 LH abscess	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
9	1937 abscess B	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
10	1938 RF milk	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
11	1938 RH abscess	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
12	1938 LF abscess	Orange to light fawn	+	+	+	+	+	+	—	—	Acid and coagulated	Died
13	1937 RF milk	Orange to light fawn	+	+	+	+	+	+	—	+	Soft curd	—
14	1932 LH milk	Orange to light fawn	+	+	+	+	+	+	—	—	Soft curd	—
15	Bottled milk	Orange to light fawn	+	—	+	+	+	+	—	+	—	—
16	1932 LF milk	Lemon yellow	+	+	+	—	+	—	+	+	Acid and coagulated	—
17	1932 LF milk	Lemon yellow	+	+	+	—	+	—	+	+	Acid and coagulated	—
18	1938 RH milk	Lemon yellow	+	+	+	+	—	—	+	+	Soft curd	—
19	1949 LH milk	Orange	+	+	—	—	+	—	—	+	—	—
20	1949 LF milk	Dirty yellow	+	+	—	—	+	+	—	—	Acid and coagulated	—

Growth in bouillon was turbid in all cases.

same clinical manifestations of disease as occur in field cases. Cow 1932 calved for the first time on October 6, 1934. Two weeks after parturition, the hair over the udder and rear parts was clipped short in order to keep the cow as clean as possible. Before each milking, the udder was washed with a sterile cloth moistened in warm water. It was next wiped with another dry sterile cloth. Later it was noticed that small abscesses occasionally appeared on the skin over the quarters. October 31, a small abscess over the posterior ventral portion of the left hind quarter was incised and culture 6 was recovered from the exudate. From October 9, the milk was examined daily for 31 days, with the result that at times the staphylococcus count varied between 10,000 and 20,000 per cc. At different intervals during these preliminary tests, staphylococci were frequently cultured and found to be of the large, non-pathogenic variety which does not attack mannite. November 16, culture 6 was introduced into the left hind quarter by means of a glass rod with a small beaded end. Approximately 800 staphylococci were conveyed to the udder on this inoculation. A second injection was made ten days later, in which about 500 staphylococci were instilled into the quarter. Between the first and second inoculations the typical microörganisms were recovered on only one examination. On December 3 (6 days later), the quarter was injected with about 200 staphylococci. A fourth inoculation with approximately 900 organisms was made four days later. From then on, a series of five inoculations, spaced at 3- or 4-day intervals, was performed. In an interval of 15 days between the third and last inoculations, typical staphylococci were recognized on six different occasions. During this period, about 2,500 staphylococci were injected. On the second day following the last inoculation, a severe acute mastitis developed. The quarter was greatly swollen, congested and painful to manipulation. The secretions were scanty, yellowish and thick. The following day the temperature was 41° C. There was inappetence with diarrhea. The cow was very sick and occasionally had a severe chill. The cell count of the milk was above 35,000,000 per cc, with high points in chlorides and pH. From then on until the cow was humanely destroyed on January 4, she progressively became emaciated and weak. From the graph it is evident that the left hind quarter was severely involved. The chloride determinations were much higher than in any case of mastitis so far encountered. On the second, third and fourth days of infection, a very high peak in the number of staphylococci occurred, which later reached a low level for four days. Thereafter the organisms

again appeared in the milk in enormous numbers. During the course of the disease, the temperature always was elevated and before death the udder felt cold to the touch.

Discussion

In the cases studied, the saprophytic staphylococci found in the udder have been separated morphologically and culturally from the staphylococci associated with mastitis. The latter could

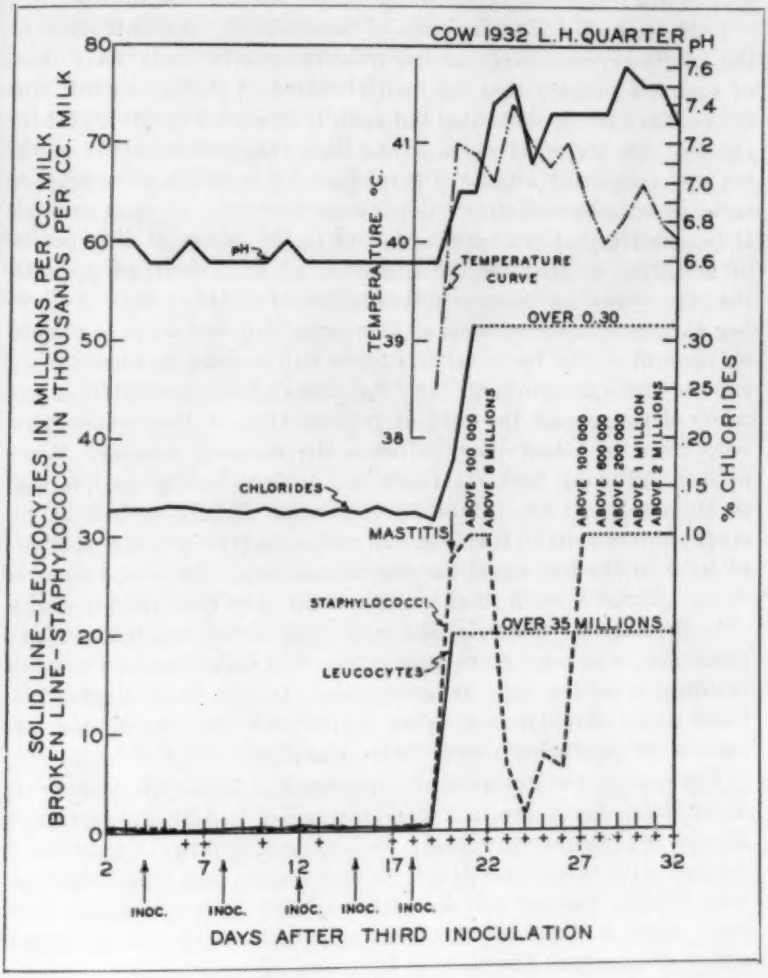

Fig. 5. Graph showing the results of the inoculation of the left hind quarter of cow 1932 with staphylococci obtained from a superficial abscess of the skin of the udder.

not be differentiated from the organisms found in abscesses of the skin of the udders.

In the normal udder, the saprophytic staphylococci may thrive in enormous numbers without causing any appreciable change in the character of the secretions. They rarely, if ever, produce mastitis. With the pathogenic staphylococci the onset of the infection is very acute, with extensive swelling of the gland. The mastitis is associated with fever and malaise, indicating a septicemia frequently terminating in the death of the animal.

It is suggested that in cases of mastitis the concentration of the inhibitory substance in the affected quarter may have been of such low potency that the multiplication of the organisms was not confined to the teat-canal but rapidly extended to the secretory tissues. On the other hand, it has been reported by other workers that frequently attacks of staphylococcic mastitis were encountered in cows immediately following parturition. In such animals it is possible that the serum passing to the udder at this period of gestation so changed the character of the colostrum or milk that the organism became well established at this time. Following parturition, the udder secretions may fail to return to normal on account of the bacterial irritation and a suitable medium for rapid growth is provided. In fatal cases or in cows with severe udder derangement the rate of introduction of the microörganisms into the udder may influence the ensuing disease. When actively growing bacteria reach the udder, multiplication may be so rapid that the inhibitory substance usually so potent for staphylococci fails to function. In udders carrying large numbers of cocci in the teat-canal the organisms may have been conveyed to the quarter in such small numbers that they were held in check.

In the skin the staphylococci may cause either small superficial abscesses or extend more deeply into the subcutaneous tissues, creating a severe local inflammation. It has been shown that these same staphylococci, when introduced into the udder, are capable of producing a very acute mastitis.

The question arises as to the incidence of udder abscesses. In many dairy herds the hair over the udder and rear quarters is clipped for sanitary purposes. Before each milking, the udder is washed with water and dried. It is possible that this procedure may remove the natural secretions of the skin which, in turn, may cause a dry, unnatural scaly condition of the epidermis which predisposes the skin to bacterial invasion.

Barber[12] and Ramsey and Tracy[13] have reported that staphylococci derived from the udders have been responsible for attacks

of gastro-enteritis in man. Further study will be necessary to determine whether the bovine staphylococci here described have cultural characters different from the food-poisoning strains.

Conclusions

It has been shown that certain staphylococci isolated from milk secretions of normal udders, from fatal cases of mastitis and from superficial abscesses of the skin of the udder, comprise a definite group possessing common characteristics. Freshly isolated strains are pathogenic for mice, and mastitis has been produced in a cow by small numbers of organisms from a culture obtained from a skin abscess.

References

[1]Guillebeau, A.: Studien über die Milchfehler und Euterentzündungen bei Rindern und Ziegen. Landw. Jahrb. Schweiz., iv (1890), p. 27.

[2]Savage, W. G.: Rep. Med. Off. Local Govt. Bd., xxxvii (1907-08), p. 425.

[3]Evans, A. C.: The bacteria of milk freshly drawn from normal udders. Jour. Inf. Dis., xviii (1916), pp. 437-476.

[4]Jones, F. S.: Studies in bovine mastitis. III. Infection of the udder with micrococci and other microörganisms. Jour. Exp. Med., xxviii (1918), pp. 721-733.

[5]Carpenter, C. M.: Experimental production of bovine mastitis with streptococci and other bacteria. Jour. Inf. Dis., xxxi (1922), p. 1.

[6]Minett, F. C., Stableforth, A. W., and Edwards, S. J.: Studies on bovine mastitis. I. The bacteriology of mastitis. Jour. Comp. Path. & Therap., xlii (1929), pp. 213-231.

[7]Plasteridge, W. N., Anderson, E. O., Brigham, G. D., and Spaulding, E. H.: The streptococci of chronic bovine mastitis. Conn. Agr. Exp. Sta. Bul. 195 (1934).

[8]Jones, F. S., and Little, R. B.: The bactericidal property of cow's milk. Jour. Exp. Med., xlv (1927), p. 319.

[9]Jones, F. S.: The bactericidal property of milk. Cert. Milk Proc. (1929), pp. 202-213.

[10]Hucker, G. J.: Studies on the coccaceae. II. A study of the general characters of the micrococci. N. Y. State Agr. Exp. Sta. Bul. 100 (1924).

[11]Breed, A. F.: Micrococci present in the normal cow's udder. N. Y. State Agr. Exp. Sta. Bul. 132 (1928).

[12]Barber, M. A.: Milk poisoning due to a type of *Staphylococcus albus* occurring in the udder of a healthy cow. Philippine Jour. Sci., Sec. B 9 (1914), p. 515.

[13]Ramsey, R. J., and Tracy, P. H.: Food poisoning probably caused by orange colored staphylococcus from udders of apparently healthy cows. Proc. Soc. Exp. Biol. & Med., xxviii (1931), p. 390.

U. S. Directory of Tuberculosis-Free Areas

The U. S. Department of Agriculture has issued a mimeographed summary showing the bovine tuberculosis situation in each state and county admitted to the so-called modified accredited area. This publication is designated as Bureau of Animal Industry Order 354, dated July 1, 1935. It contains in each case the date on which the tuberculosis-free status of a county expires, until extended by evidence that infection is still less than one-half per cent. Amendments are to be issued monthly to keep this information up to date.

IMMUNIZATION AGAINST VIRUS DISEASES WITH TISSUE VACCINE*

By WILLIAM HUTCHINS BOYNTON, *Berkeley, Calif.*
Division of Veterinary Science, University of California

INTRODUCTION

In discussing the properties common to tissue vaccines, the title presented to the writer affords him, at the very outset, the opportunity to emphasize the fallacy of overestimating the efficacy of tissue vaccines as they are prepared and administered at the present time. To develop a solid, long-lasting immunity, an animal must either suffer an attack of a disease caused by a virus or must be given potent virus accompanied by a protective agent, such as serum from a recently-recovered animal or from one which has been hyperimmunized against the disease in question. In either case, the immunity produced is a result of a more or less severe reaction to the virus and, therefore, tends to be lasting, except in the case of young animals which may tend to outgrow their immunity. In respect to tissue vaccines, resistance is, perhaps, a more fitting term than immunity. Properly prepared and administered, tissue vaccines do produce a high grade of resistance, varying from one to several months and possibly one or more years, depending upon the age, health and ability of the animal to respond to the vaccine. In rinderpest, hog cholera, dog distemper undoubtedly, and possibly other virus diseases, it must be remembered that young, suckling animals either rapidly outgrow any resistance they may develop through vaccination, or, if they are suckling immune mothers, do not develop antibodies readily at all. The best results are obtained, then, from mature or, at least, weaned animals.

Another rather common fallacy is to consider one dose of a tissue vaccine sufficient to protect an animal against a virus disease. This belief is responsible for many "breaks." It is a far wiser procedure to use two or more injections to provide adequate protection.

The purely prophylactic character of tissue vaccines limits their use. They are not therapeutic in any sense and, therefore, are of no benefit to a sick animal or to one in the incubation period of a virus disease.

---

*Presented at the seventy-second annual meeting of the American Veterinary Medical Association, August 27-30, 1935, Oklahoma City, Okla.

Requirements for a Tissue Vaccine

To prepare any tissue vaccine, there are two requisites. First, the animal from which the tissues are to be obtained should be sacrificed at the height of the disease. In rinderpest and hog cholera, this period is approximately the third day after the initial rise in temperature. If the disease is allowed to progress too far, there is the possibility of formation of antibodies, or development of secondary complications in the animal, both of which lessen the strength of the virus. It is of the greatest importance to obtain tissues as saturated as possible with potent virus.

The second requisite is the use of those tissues which contain virus in highest concentration. After many experiments, these have been found to be the organs and tissues rich in reticulo-endothelial cells; namely, the spleen, lymphatic system, glandular tissue, and red marrow of bones. Although the blood is virulent in these diseases, it is, undoubtedly, merely a vehicle for carrying away the excess of virus shed from the above-mentioned tissues, and not a medium in which the virus multiplies to any great extent. Observation has shown that it, as well as the heart, liver and kidneys are not suitable tissues, except that when added to the above-mentioned tissues they increase the yield of vaccine.

In the tissue vaccine, the virus must be so modified by chemical, physical or mechanical means that it loses the power of producing disease, but retains the ability to stimulate antibody formation in the animal body. Since viruses respond differently to attenuating agents, various means of modification must be tried for each particular disease until a suitable method is found.

There is considerable controversy as to whether the virus thus modified is alive or dead. It is the writer's opinion, after a number of years of observation and experimentation, that killed virus has no protective power. It has been demonstrated that if a high percentage of a chemical attenuating agent, such as phenol, chloroform, formalin, or toluol, be added to rinderpest, or eucalyptol to hog cholera vaccines, these will have no protective value. If a moderate content of one of these agents be present, the vaccine will be rapidly attenuated sufficiently and will develop adequate resistance, but the vaccine loses its potency in a month or two. If but a small amount of chemical be added to the vaccine, some time must elapse before the virus is sufficiently modified, but the vaccine remains potent for several months. In rinderpest, for example, if vaccines attenuated by phenol and heating were kept at 41.5° C. for three hours, it was necessary

to complete the attenuation by storing them in the refrigerator for some time before they were safe for use, but such vaccines did retain their potency for several months. Vaccines heated at 43.5° C. were more rapidly attenuated, but lost potency more quickly and required larger doses for effective vaccination. Heating to between 44 and 45° C. destroyed the protective power of the vaccine completely. Other experiments showed that, after 48 hours at room temperature, vaccine began to lose its value and was no longer useful after 96 hours, although portions of the same vaccine, stored in the ice-box, would retain their potency for several months.

If, then, a tissue vaccine is composed of a dead virus, why do these changes in potency occur? Do they not indicate rather the modifying effects exercised by an attenuating agent upon a live virus?

Application of the Tissue Vaccine

Rinderpest: The eradication of rinderpest from the Philippines is a classic example of what may be accomplished by the use of tissue vaccine, and for that reason a brief history of this achievement will be given here. (The author has no desire to detract from the splendid work done by investigators on rinderpest in other countries, but, for brevity, confines his remarks to the Philippines because his own investigations were carried on there.)

According to Youngberg,[1] it is generally believed that rinderpest was introduced into the Philippines in 1886 or 1887, through the importation from French Indo-China of carabaos intended for breeding purposes. The disease spread extensively, causing enormous losses of cattle and carabaos; the mortality in some localities reached 90 per cent. Severe epizoötics came in cycles of from eight to ten years. The first wave was in 1887; the second, about 1897; the third, 1907; and a decided increase in the incidence, if not an epizoötic, occurred in 1916.

Over this period of years, numerous investigations were carried out in the hope of controlling the disease. About 1901, the glycerin-bile method was tried unsuccessfully. In 1902, a serum laboratory was established. Various workers, including Ruediger,[2] Thomson,[3] Ward and Wood,[4] Topacio,[5] Kern,[6] Youngberg and Shaffer,[7] and the writer[7] added considerably to the store of knowledge concerning rinderpest antiserum and reduced the cost of serum production to some extent. Good results were obtained with the simultaneous method in districts where the disease was purely enzoötic; however, in rinderpest-free areas or in localities where

the infection has been recently introduced, its use was dangerous because the immunization stations were a constant focus of infection. Treatment with serum alone was both expensive and inefficient, since the protection it afforded lasted from ten days to three weeks only, after which period, the animals were susceptible to re-infection. The quarantine and slaughter method was used with some success by Thomson[8] in Davao, but failed in other provinces, due chiefly to lack of coöperation on the part of owners, who, refusing to have their sick and exposed animals slaughtered, would not report the disease, would hide the sick from the inspectors, and slip the exposed animals through the quarantine lines at night. After the extensive experiment, on transmission by Ward, Wood and the author,[9] indicated that virus did not survive long in the field, quarantine without slaughter was used largely. However, when the quarantine was lifted, animals not having undergone the disease were as susceptible as ever, and in many cases, also, re-infection would occur. Observations of Youngberg[10] and experimental work of the writer[10, 11] showed that swine and a large water leech widely prevalent in carabao wallows could transmit the disease. All of the above-mentioned factors made these methods of control unsatisfactory both to cattle-owners and to the veterinarians in charge.

In 1914, Kakizaki[12] developed the first tissue vaccine for rinderpest, using toluol as the attenuating agent. Three years later, the writer, unfamiliar with the Japanese literature, observed independently the possibility of using such a vaccine. A brief summary of the experiment follows:

On February 12, 1917, carabao 73, in its second day of temperature, was bled to death. Two hundred and fifty grams of its finely-ground liver was placed in 500 cc of 0.5 per cent phenol solution and 200 gm of finely-ground lymph-glands was placed in 400 cc of 0.5 per cent phenol solution. These mixtures then were placed in the ice-box and agitated at frequent intervals during the following two days. On February 14, the extracts thus produced were filtered through filter paper in the ice-box overnight. On the following day, the completely-filtered extracts were bottled and stored in the refrigerator. Twelve days later, carabao 79 was inoculated subcutaneously with 100 cc of liver extract and the same amount of lymph-gland extract. No ill effects were noted in the animal as a result of this massive injection.

To test the resistance of the animal to rinderpest, on March 17, 30 days after vaccination, carabao 79 received 50 cc of virulent blood from a sick carabao (85) and was placed in the stall which

had been occupied by 85. Carabao 79 showed no symptoms of the disease.

From this date, experiments were begun to develop a safe, reliable vaccine. Various changes and improvements in technic were made until the vaccine proved ready for the field.[13] It then consisted of a concentrated mixture of blood and finely-ground tissues from the spleen, liver, kidney, lymph-glands, heart and testicles, to which were added one-third its volume of glycerin (pH 7.8) and 0.5 per cent phenol. This mixture was heated at 42° C. for three hours and, just before its use, was diluted with a fluid consisting of 33 1/3 per cent of glycerin in physiological saline.

Through the enthusiasm and foresight of the late Dr. Ildefonso Patdu, who was able to interest the native cattle-owners in this tissue vaccine, the province of Rizal was freed from rinderpest by 1921. At the same time the mortality in other parts of the Philippines was recorded as 35.740. In the following year, the use of rinderpest vaccine was commenced in the field on a large scale.

Personal communication from Doctor Youngberg in October, 1926, stated that by January 1, 1927, over 300,000 head of cattle and carabaos would have received vaccine. About this time Kelser, Youngberg and Topacio[14] introduced modifications in the preparation of the vaccine, using spleen, liver and lymph-glands (except the mesenteric) in physiological saline and adding 0.75 per cent of chloroform instead of phenol as the attenuating agent. During 1929, the vaccine was still further improved[15] by using spleen, lymph-glands and tonsils, and reducing the chloroform content to 0.375 per cent. In place of the two or three subcutaneous injections given formerly, Rodier[16] showed that a single intramuscular injection was sufficient to provide resistance for a few months. The newly-modified method was superior to the old method, since, with the period for attenuation now shortened by the substitution of chloroform for phenol, the vaccine retained its potency for some months and afforded greater protection.

The methods described necessitated refrigeration and this involved transportation difficulty. To overcome this obstacle, a dried vaccine was prepared by Robles and Generoso,[17] similar to that prepared by the old method of phenol attenuation except that no diluting fluid was used. The material was dried over calcium chloride and kept in the ice-chest at 0 to 5° C. during the drying process. This was found to retain its potency for 20 days at atmospheric temperature.

The results of the application of these various modifications of the tissue vaccine are significant when one compares the 629,176 estimated fatalities of the years 1901 and 1902 with the 50 recorded deaths from January to June, 1932. One small outbreak[18] occurred in a municipality in the province of Isabela during April, 1934, and since then, as far as is known, no outbreak has been discovered in the entire archipelago.

The successful control of rinderpest in the Philippines is due not merely to mass vaccination. Other contributory factors are the prohibition of importations of cattle and carabaos from infected countries, quarantine, and constant surveillance. Great credit is due Dr. Archibald R. Ward and his successors, Dr. Stanton Youngberg and Dr. Victor Buencamino, for keeping all measures for the eradication of rinderpest in the hands of veterinarians.

Hog cholera: In contrast to the method of handling rinderpest in the Philippines, no such supervision is provided for the control of hog cholera in the United States. In most states, anyone may vaccinate his pigs; consequently, with the serum-virus method now in use, the disease is still wide-spread. The use of virus in the field is dangerous enough in the hands of veterinarians; in the hands of laymen, it is disastrous.

The immunization of swine against hog cholera by the simultaneous method is, in itself, easily accomplished and very successful, providing the animals vaccinated are healthy and receive proper food in not too large quantities during the period in which their immunity is being built up. The protection thus acquired is stronger and of longer duration than that provided by the tissue vaccine method; however, swine treated by the serum-virus method will, while undergoing their reaction to serum-virus, in many instances, readily transmit hog cholera to susceptible animals, but swine treated by the tissue vaccine method may cohabit with susceptible animals without the slightest danger of transmitting the disease.

Hog cholera immunization offers a greater problem than rinderpest because it is so frequently complicated with the secondary infection called necrotic enteritis. Undoubtedly, swine infected with *Salmonella suipestifer, Ascaris,* or, for other reasons, unthrifty, do not readily develop resistance to hog cholera by any method of vaccination, but they do not suffer any ill effects from the administration of the tissue vaccine. In the simultaneous method, however, the reaction of the animal body to the presence of hog cholera virus tends to bring about a temporary lowering of the general resistance and this furnishes an oppor-

tunity for other conditions to develop; in other words, "breaks" occur. This is particularly true with respect to necrotic enteritis. To cite an example, on one ranch, with which the writer has been in contact, the estimated loss from enteritis following vaccination with serum and virus was 25 to 60 per cent. This is, perhaps, an extreme case, but the losses generally are much too large. Whether or not any resistance to hog cholera is developed by either method under unfavorable conditions, at least the pig stands a better chance of reaching market when the tissue vaccine method is used, since he all too frequently succumbs to secondary infection when the serum-virus method is used.

In this connection, mention may be made here of some work of the author and his assistant now in progress. Field trials are being made to immunize very young pigs against *Salmonella suipestifer* by means of a bacterin in order that they may be able to withstand any flare-up of the infection following vaccination against hog cholera. Some success has been attained but the investigation has not been carried far enough to warrant any conclusions as yet.

Feed is another factor to be reckoned with in the simultaneous method. If the feeds to which the swine are accustomed are changed during the time the animals are reacting to the serum and virus, serious trouble may be anticipated. Since it is harmful also to allow the animals to eat too much at this time, the rations should be lessened a little. In the tissue vaccine method, the absence of unmodified virus makes the problem of feed negligible.

The tissue vaccine method described by the writer[19] in a previous report has been improved with slight modifications and, briefly, consists of three volumes of finely-ground tissues from the spleen, lymph-glands, kidneys, testicles, and red marrow of bones mixed with two volumes of buffered glycerin (pH 7.6) and 0.5 per cent eucalyptol. The mixture is ground in a ball mill at approximately 6° C. for 60 hours, then is strained through a sieve and bottled for storage in the refrigerator.

After preservation for 25 to 30 days, this vaccine is administered in two injections at weekly intervals of 5- or 10-cc doses intramuscularly or of a combination dose of 2 cc intramuscularly and 1 cc intradermally. With swine kept under experimental conditions, excellent results have been obtained. Hogs have been found still resistant to the virus eleven months after vaccination. How much longer the resistance lasts has not yet been determined.

Hog cholera vaccine was shipped to Ames, Iowa, to be tested at the Bureau of Animal Industry Experiment Station there.

A report from the late Doctor Dorset, dated October 20, 1934, is quoted:

> In all, eight pigs were treated with the vaccine. Four of these received a single injection of 10 cc in the muscle of the neck and four received two injections one week apart, 10 cc for each injection, on opposite sides of the body. These pigs were held three weeks after the last vaccine injection and then were exposed by the injection of 1 cc of virus administered subcutaneously. Following this virus exposure none of the vaccinated pigs became visibly sick, although of four controls, injected at the same time, three contracted hog cholera while the fourth remained well. It appears, therefore, that the vaccine gave adequate protection to the vaccinated pigs.

With this report to recommend it, the tissue vaccine is now considered to be about ready for field application.

Canine distemper: The virus of canine distemper is similar to that of rinderpest in that exposure to external conditions rapidly destroys it. The disease, however, resembles hog cholera in having associated with it chiefly, among other organisms, the secondary invaders, paratyphoid A and the organism classified by Bergey as *Alkaligenes bronchisepticus*. If the secondary infection could be controlled, the serum-virus method would be a simpler procedure, but, under present conditions, that type of vaccination is fraught with the same danger as in hog cholera.

During the early part of 1927, the writer prepared two lots of vaccine by the same technic used for rinderpest except that 0.5 per cent toluol was substituted for phenol in one lot. The puppies tested appeared to be successfully vaccinated in both cases, but the publication of the Laidlaw-Dunkin method about that time caused the work to be discontinued.

Dr. F. W. Wood, of the Cutter Laboratory, applying the Laidlaw-Dunkin method, claims a safe and long-lasting resistance may be developed by giving, at fortnightly intervals, two inoculations of a 5-cc dose distributed over at least two places on the body, and following these two injections with a third inoculation two months later.

Some workers advocate the use of canine distemper vaccine supplemented with virus injection later. The method is excellent for building up immunity, but the same objection holds for this as for any method involving the use of unmodified virus. The purpose of vaccination is defeated with the introduction of virus to the field, resulting in the probability of setting up fresh foci of infection.

SUMMARY

In summing up the advantages of each method of combating virus diseases, the writer would stress once more the fact that

the simultaneous method produces a superior type of immunity. The tissue vaccine is limited in that it provides a less adequate immunity and has no therapeutic properties; however, it is infinitely safer, since it eliminates unmodified virus from the field and renders negligible the dangers of giving rise to secondary complications. Properly controlled, by keeping the tissue vaccine in the hands of veterinarians who know when and how to use it, such a vaccine should safely control losses from virus infections and go far toward eradicating virus scourges in which methods involving the use of unmodified viruses have thus far failed.

References

[1]Youngberg, S.: A brief history of rinderpest in the Philippine Islands. Philippine Agr. Rev., xv (1922), 3, p. 205.
[2]Ruediger, E. H.: Filtration experiments with the virus of cattle plague. Philippine Jour. Sci., iii (1908), p. 165.
[3]Thomson, C. G.: Anti-rinderpest serum, its production and use. Philippine Agr. Rev., ii (1909), pp. 672-673.
[4]Ward, A. R., and Wood, F. W.: Experiments on the efficiency of anti-rinderpest serum. Bul. Philippine Bur. Agr. 19 (1912).
[5]Topacio, T.: The manufactory of anti-rinderpest serum in the Philippine Islands. Philippine Agr. Rev., xv (1922), 3, pp. 229-236.
[6]Kern, H. F.: The use of anti-rinderpest serum in the field. Philippine Agr. Rev., xv (1922), 3, pp. 241-245.
[7]Boynton, W. H.: Note on the use of organ extracts in place of virulent blood in immunization and hyperimmunization against rinderpest. Philippine Jour. Sci., xiii (1918), p. 152.
[8]Thomson, C. G.: Report on the recent outbreak of rinderpest in the district of Davao, Mindanao. Philippine Agr. Rev., iv (1911), 5, pp. 243-247.
[9]Ward, A. R., Wood, F. W., and Boynton, W. H.: Experiments upon the transmission of rinderpest. Philippine Jour. Sci., ix (1914), pp. 49-79; Bul. Philippine Bur. Agr. 30.
[10]Boynton, W. H.: Rinderpest in swine with experiments upon its transmission from cattle and carabao to swine and vice versa. Philippine Jour. Sci., xi (1916), 5, pp. 215-263; Philippine Agr. Rev., ix (1916), p. 288.
[11]Boynton, W. H.: Duration of the infectiveness of virulent rinderpest blood in the water leech, *Hirudo boyntoni* Wharton. Philippine Jour. Sci., viii (1913), pp. 509-521.
[12]Kakizaki, C., Nakanishi, S., and Oizumi, T.: Experimental studies on prophylactic inoculation against rinderpest. Report III, Jour. Jap. Soc. Vet. Med., v (1926), 4, pp. 221-276.
[13]Boynton, W. H.: Rinderpest with special reference to its control by a new method of prophylactic treatment. Philippine Jour. Sci., xxxvi (1928), 1, pp. 1-35.
[14]Kelser, R. A., Youngberg, S., and Topacio, T.: An improved vaccine for immunization against rinderpest. Philippine Jour. Sci., xxxvi (1928), 4, pp. 373-393.
[15]Robles, M. M.: An evaluation of the data on rinderpest vaccine in the Philippines. The Gazette, Philippine Dept. of Agr., ii (1932), 8, pp. 5-9.
[16]Rodier, E. A.: A single-injection method of immunization against rinderpest. Philippine Jour. Sci., xxxvi (1928), 4, pp. 397-407.
[17]Robles, M. M., and Generoso, J. D.: Dried rinderpest vaccine. Philippine Jour. of Animal Ind., iv (1934), 1, pp. 33-42.
[18]Buencamino, V.: The job is not yet finished. Philippine Jour. of Animal Ind., i (1934), 4, pp. 183-184.
[19]Boynton, W. H.: A tissue vaccine for hog cholera. Jour. A. V. M. A., lxxxiii (1933), n. s. 36 (6), pp. 747-763.

Discussion

Dr. B. T. Simms: I would like to ask Dr. Boynton concerning the systemic reaction that he observed. We discussed that subject in Chicago two years ago, and I believe some charts that he showed us indicated that there was a temperature rise, rather a slight but nevertheless a noticeable temperature rise, in from six to eight days following the injection of tissue vaccine; and that he did not get such a systemic reaction at a later period when he injected virus. I am

wondering if, with the changes that he has made in the method of preparation, he still sees this slight systemic reaction following the injection of the tissue vaccine.

LT. COL. R. A. KELSER: This whole question of tissue vaccines in connection with virus diseases is exceedingly important. Such type of vaccine will probably constitute the only means that we will have for some years, to immunize against virus diseases. Not until we are able to culture viruses artificially in large quantities will we be able to get away from the use of tissue vaccines.

There is another thought that has often presented itself to me in connection with the study of viruses, which might indicate that even though we are able to grow viruses artificially, we might still not be able to get away from the tissue vaccine in the production of immunity. There have been, time and again, instances which might well be interpreted as indicating that immunity produced by these so-called tissue vaccines may not be due to their content of dead virus, or modified living virus. It may be due to such virus alone, or it may be due to a combination of virus, plus products of reaction between virus and tissues. Such a possibility has suggested itself in rabies immunization studies and also in connection with yellow fever.

As Dr. Boynton pointed out, there has been considerable discussion as to whether or not the virus in these vaccines is actually dead. That is a debatable question, and opinion is considerably divided. He is of the opinion that a dead virus will not immunize against a subsequent dose of a live virus. I am more inclined to believe that the question involves a quantitative factor. I believe that ordinarily a small dose of dead virus will not immunize against a subsequent dose of the same virus in a live state. On the other hand, if you have a sufficient quantity of dead virus present in the vaccine, I think it will immunize. In other words, we may get the same result with a small amount of live virus, or a large amount of dead virus. I have come to the conclusion, personally, that you do get immunity from dead virus if you have enough of it.

Dr. Boynton also mentioned the value of repeated doses over one large dose. I am thoroughly in accord with that. I feel that the sum total of the immunity resulting from several distinct and separate reactions following injections of these tissue vaccines is greater than that following the same quantity of vaccine given in a single dose.

The question of the preparation of these tissue vaccines is not so easy, as might be inferred from Dr. Boynton's discussion. For instance, with rinderpest vaccine, prepared according to his phenol-glycerin-heat method, it is not possible for one to prepare lot after lot of the vaccine uniformly, according to the method described, and have each successive lot ready for use in approximately the same number of days, or have it potent when it is safe to use. The same thing is probably true of other tissue vaccines, but we can not measure the potency of them so accurately as we can that of rinderpest vaccine.

I think that, all in all, until we learn more about viruses generally, from an immunization standpoint the field is going to be tissue vaccines.

DR. FRANK BREED: Col. Kelser, relative to your phenol virus, is there not a possibility that you might have two radicals—one a disease-producing radical and one an immunity-producing radical?

LT. COL. KELSER: There is considerable question as to just what is the precise stimulating agent in artificially induced immunity to these various virus diseases in which tissue vaccines are used. One of several possibilities is that you may have various stages or divisions of the virus, and that you might have one stage in the blood or humors and another intracellular. Now as to the other thing that Dr. Boynton

suggested—that treatment might not actually kill the virus biologically. I know of no accurate test for biological death of a virus. An animal is inoculated and if it survives and does not develop disease, the virus is commonly considered dead. The question as to whether or not it is definitely biologically dead, however, would depend on some means of being able to prove by artificial growth that you have actually destroyed the virus. While to all intents and purposes a virus appears to be dead, until we get some test of that sort, one can not positively prove or disprove that the thing producing immunity is dead.

DR. BOYNTON: Dr. Simms, in many instances we still observe a slight reaction in vaccinated animals. This reaction, which may be a slight rise in temperature, usually occurs one or two days following vaccination and subsides in a day or two. Since, in the case of hog cholera, susceptible animals may be kept in close contact with vaccinated ones without contracting the disease, this reaction to the vaccine is probably not an indication of developing disease, but is rather, in my opinion, a response of the animal body to a protein injection. I am of the opinion also that this reaction may stimulate antibody formation and thus be beneficial, although I have no evidence on which to base this statement.

Cattle and Swine Population

According to a recent estimate made by the Imperial Economic Committee, the cattle population of the world is approximately 600,000,000. The number of hogs is estimated at about 300,000,000; and sheep at about 750,000,000. Meat consumption in the United States and Great Britain is estimated at 150 pounds annually per person; in Germany at 110 pounds, and in France at 90 pounds.

Object to Ban on Horse Traffic

Resolutions of protest against a proposal to ban horse traffic in the streets of large towns in Great Britain have been transmitted to the Ministry of Transport by the National Horse Association, the Clydesdale and Shire Horse societies and many other British organizations, including chambers of commerce, agricultural societies and private trading concerns.

Department of Agriculture Yearbook

The Yearbook of the U. S. Department of Agriculture for 1935, now available, contains articles on practically all phases of the technical and economic research that is carried on in the Department. Among the topics discussed are the eradication and control of insect pests, animal husbandry, forest husbandry, chemical investigations and soil erosion. The volume contains also the annual report of the Secretary of Agriculture for 1934.

EXPERIMENTS LEADING TO AN EFFECTIVE TREATMENT FOR CANINE WHIPWORMS*

By H. M. CORENZWIT, *Philadelphia, Pa.*

LIFE HISTORY

The adult whipworm *(Trichuris depressiusculus)* resides in the cecum of the dog. The ova passing out with the feces infest the premises. There is no intermediate host or free-living stage. The ova embryonate quickly in warm weather and slowly in cold weather. The dog becomes infested by licking the paws or other objects that are contaminated. In the alimentary tract of the dog, the embryos emerge from the egg-capsules and enter the glands of Lieberkühn where they stay for three or four days. The larvae are reported found in the cecum five days after ingestion of the embryonated ova. Some authors state that sexual maturity is reached in two to four weeks; others, that several months are required.

Wright[1] reported finding 81 per cent of 150 dogs taken from the Washington, D. C., pound infested with whipworms. The number of specimens found in the individual dog varied from one to 738.

SYMPTOMS AND DIAGNOSIS

There are no pathognomonic symptoms. In some cases symptoms are absent or there may be symptoms simulating those of chronic intestinal catarrh. Principally noted are skin, ear and eye conditions, lassitude and occasional diarrhea.

Diagnosis is based on the microscopic examination of the feces and finding the characteristic lemon-shaped ova.

PATHOLOGY

Necropsies would seem to indicate that whipworms are of little pathologic importance, as the cecum, even in heavy infestations, appears normal macroscopically. However, occasionally severe inflammation of the cecum and adjacent parts of the ileum and colon is noted. To the writer it appears that this parasite causes distress to a degree that is out of proportion to the number of worms present and the local lesions found on necropsy. Promptly improved health follows the medicinal removal of the worms or the surgical removal of the infested cecum, in unhealthy dogs showing the constant presence of numerous whip-

*Demonstrated at the clinic held in conjunction with the seventy-second annual meeting of the American Veterinary Medical Association, Oklahoma City, Okla., August 27-30, 1935.

worm ova in the feces. Whipworms probably do their greatest harm through enteric absorption, by the host, of a toxic excretion rather than by any mechanical injury. The body response may be of an allergic nature.

Treatment in Vogue

Drugs per os: Santonin, tetrachlorethylene, oil of chenopodium, and other drugs are given *per os* before breakfast for several consecutive days. Results generally are poor. Ova usually continue to pass and any improvement noted is probably due to concurrent treatment. Leche de Higueron, a drug derived from a South American fig tree, is of value in human trichuriasis, but the author has seen no work recorded on dogs in which the drug was used.

Cecectomy: Results of cecectomy are excellent, but for several obvious reasons clients do not readily consent to this operation.

Colonic injection of drugs: This method of treatment, employing the colon-tube, is discussed in detail in this paper.

History of Colonic Injection Treatment

Underwood, Wright and Bozicevich[2] reported the passage of a No. 30 Davol catheter 17 inches into the colon of a dog, followed by the injection of 10 cc of a 2 per cent solution of mercurochrome. An immediate autopsy showed the cecum full of the solution. In another dog, the tube was passed 14 inches and the proximal half-inch of the cecum was found stained. Six infested dogs were thus treated with 10 cc of tetrachlorethylene in 90 cc of water. The results were: One dog passed whipworms; none found on autopsy. Five dogs passed no whipworms; whipworms found on autopsy.

In another dog a No. 30 colon-tube was inserted until resistance and uneasiness indicated that the tube would go no further. On immediate destruction and autopsy of the dog, it was found that 12 inches of tube lay in the colon with the cecum 2½ inches beyond the end of the tube. Using eight dogs, a No. 36 tube was passed as far as possible and oil of chenopodium in mineral oil or ethylidene chloride in water was injected. The results, checked by autopsies five days later, were: One dog passed one whipworm; 31 whipworms found in cecum. Six dogs passed no whipworms; whipworms found in cecum. One dog passed no whipworms; none found in cecum.

Steinbach[3] used a 9-inch, blunt-nozzled syringe on small dogs and a turkey gizzard-catheter on large dogs. These were passed into the colon. He injected one drop of oil of chenopodium per

pound of body weight in a vehicle of mineral oil. Steinbach reported complete success, with only one case requiring a second treatment. He concluded, from the absence of ova, that all whipworms were removed. Later he[4] cited cases requiring seven or eight injections. Still later he[5] reported one dog requiring 13 injections.

Anatomy Involved

It is helpful, for a better understanding of the work done, to review certain anatomical features. The large intestine is that part of the digestive tract from the ileo-colic valve to the anus. It is divided into the cecum, colon and rectum.

The cecum is a short, spirally-twisted tube which may be regarded as a diverticulum of the colon. It lies in the right flank and varies in length from 1 to 8 inches according to the size of the dog. The blind end is posterior. The attached anterior end has a single opening, the ceco-colic orifice. This orifice communicates with the beginning of the colon. The cecum is rather closely applied to the ileum by peritoneum.

The colon runs forward from the ileo-colic valve on the right of the median line, until it reaches the pyloric part of the stomach (ascending colon). Here it turns to the left and crosses the median line, bounded anteriorly by the stomach and posteriorly by the mesenteric root (transverse colon). It then bends again and passes back as a fairly straight tube, to the left of the median line (descending colon). Near the pelvis, a gradual bend to the right carries the colon to the median line where it enters the pelvis and becomes the rectum, which terminates at the anus. The colon is suspended in the short mesentery. Of paramount importance is the fact that the portion of the short mesentery which is attached to the ileo-colic juncture, and thus also controls the movability of the applied cecum, is the shortest portion of this mesenteric fold. Thus the cecum is held fairly close to the mesenteric root while the colon is easily movable.

Experimental Colon-Tube Passage

Experimental colon-tube passage was done on 27 fresh cadavers and experiment dogs. The data of seven of these dogs are compiled in table I. With the right flank cut to form a flap which could be raised for observation and dropped back to the normal position again, it was seen that the tube-end passed easily forward to a point about the center of the posterior surface of the stomach. Here, at the first bend in the colon, resistance was met. Advancing the tube resulted in a bellying of the descending colon, usually to the left. The tube-end usually would move on to a

point at or near the ceco-colic orifice. Resistance greatly increased, accompanied by the formation of one or more coils in the tube and the enveloping colon.

Occasionally sharp S-shaped bends appeared in the tube, although the enveloping colon would be straight. Manipulation through the belly wall was helpful. It was found that the factors governing the filling of the cecum with liquids by means of a colon-tube are:

Fasting: A full stomach pushes the left or descending colon to the right, producing an acute angle instead of a transverse portion. This increases resistance to the tube passage. Palpation and manipulation of the tube are easier in the fasted subject.

Enematization: Feces in the cecum prevent the entrance of liquid into all or at least the distal end of the cecum (table I, dogs 1 and 4). Feces in the colon interfere with tube passage.

Type of colon-tube: Too heavy or too stiff a tube encounters great resistance. Since the cecum is held close to the mesenteric root and is limited in its movability, too heavy or stiff a tube carries the ascending colon further from the median line. This causes an artificial bend to be formed in the first few inches of the ascending colon, with the cecum medial to the tube-end. It is impossible for a stiff tube to make this last artificial bend (table I, dog 5). Also, if too soft a tube is used, coiling of the tube occurs, with no advancement of the tube-end.

Quantity injected: In small dogs the cecum may hold less than a dram. In large dogs it may hold more than an ounce. Quantities greater than the cecal capacity naturally are lost into the colon (table I, dogs 3, 4 and 7). The bulk of any injected liquid soon passes from the cecum.

Tonus of ileo-colic valve: This is sufficient to cause injected liquids to pass first into the cecum. The valve is permeated by fluid only if the injection is made after death (table I, dog 3) or if a large quantity is injected under pressure, as in a high enema.

Mucus, air and gas in the cecum: These may prevent the passage of liquids to the tip end of the cecum where most of the whipworms are. The spirally twisted condition of the cecum favors the retention of gas bubbles driven ahead of the column of injected liquid.

Spastic condition of the cecal tip: This condition is noted with the mucosa closely applied to the opposing mucosa and the lumen only a potential one. This protects the parasites from vermicidal action.

TABLE I—*Experimental colon-tube passage.*

DOG	WT. (LBS.)	LENGTH* (IN.)	FAST (HRS.)	ENEMA	SIZE OF TUBE	INCHES PASSED	MATERIAL INJECTED	AUTOPSY FINDINGS
1	45	36	36	—	#32	21	1 ounce methylene blue solution. Tube removed and dog destroyed	Cecum packed with feces. Proximal half of cecum stained. Four inches of colon stained
2	7	22	36	—	#28	15	None	End of tube at ceco-colic orifice
3	26	31	72	—	#28	22	Dog destroyed, flank excised and then 2 ounces methylene blue solution injected	Tube end at ceco-colic orifice. Cecum entirely filled with solution. 12 inches of colon and 8 inches of ileum stained
4	5	21	None	—	#28	14	2 ounces methylene blue solution injected and dog destroyed	Tube end at ceco-colic orifice. Cecum full of feces. Proximal end of cecum slightly stained. Solution passed through colon and out of anus
5	45	39	None	+	#39	29	None	End of tube 3 inches short of ceco-colic orifice
6	10	25	None	—	#28	16¾	None	End of tube at ceco-colic orifice
7	?	33	None	+	#28	21¼	2 ounces methylene blue solution injected and dog destroyed	End of tube at ceco-colic orifice. Cecum filled, colon heavily stained

Anesthesia: This prevents struggling and straining and favors manipulation and palpation through the belly walls.

It seemed reasonable to believe that in the great majority of cases the cecum could be filled with a vermicidal liquid if care was observed. The distance necessary to pass the tube was about two-thirds of the body length from the anus to the tip of the outstretched nose. Body weight was not a factor.

Experimental Treatment of Trichuris-Infested Dogs

Ten dogs (28 to 37, table II) were selected for experimental treatment by colon-tube. A No. 28 Davol tube was used. The findings are recorded in table II.

Table II—*Findings in experimental treatments for whipworms.*

Dogs	Vermicide	Results	Later Findings
28-33	A mixture of oil of chenopodium, arecolin, crotin oil, santonin, oil of turpentine and tetrachlorethylene in mineral oil	1 dog passed whipworms	Ova
		4 dogs passed no whipworms	Ova
		1 dog passed whipworms	Live worms on autopsy
34	Oil of chenopodium in mineral oil	1 dog passed whipworms	Ova
35	Nicotine in water	1 dog passed whipworms	Negative fecals
36		1 dog passed no whipworms	Ova
37	Nicotine in mineral oil	1 dog passed whipworms	Live worms on autopsy

As only five of the ten dogs treated passed whipworms and as four of these five continued to pass ova or showed live cecal worms on autopsy, it was decided to hunt out the factors preventing the death of the entire parasitic colony. Trichuricidal tests, nicotine toxicity tests and various technics were undertaken.

Trichuricidal Tests

Portions of excised ceca, with living whipworms attached, were placed in vessels containing various drugs. Their ranking, according to their trichuricidal efficacy, was:

1. Nicotine (0.7 grain) in water (1 ounce).
2. Ether (1 dram) in olive oil (1 ounce).
3. Tetrachlorethylene (2.5 cc) in mineral oil (1 ounce).
4. Oil of chenopodium (½ dram) in mineral oil (1 ounce).
5. Santonin (1 grain) in olive oil (1 ounce).
6. Arecolin hydrobromide (½ grain) in water (1 ounce).
7. Water at 115 to 120° F.
8. Hexylresorcinol S.T. 37 (1 ounce).

Ether in olive oil caused the worms to relax and drop off the mucosa but did not kill so quickly as nicotine in solution. Water at 115 to 120° F. had the same effect as ether. With all other drugs the worms clung to the mucosa for a considerable time even though dead. Viability was shown by movement when the parasite was placed close to a hot electric-light bulb. Hexylresorcinol S.T. 37 had no trichuricidal action in this experiment.

Nicotine Toxicity

As we feared that nicotine might have a severe toxic effect, at first, other substances were injected into infested dogs. Results were indifferent. Four experiment dogs (35, 36, 37 and 38, table III) were given large doses of nicotine in solution, three by rubber colon-tube and one by a straight glass tube, to note the toxic effect. The results of these toxicity tests are given in table III. The results showed that the work could be continued safely by using a solution of 0.7 grain of nicotine in one ounce of water. One Lilly Pulvule No. 142 in one ounce of water gives this nicotine value. The dosage to be used was decided on as follows: Small dogs, ½ ounce; medium-sized dogs, 1 ounce; large dogs, 1 to 2 ounces.

Table III—*Tests of effects of nicotine on health of dogs.*

Dog	Weight (Lbs.)	Dosage of Nicotine	Solution (%)	Dosage per Pound Body Weight (Gr.)	Result
35	30	4.2 grains in 1 ounce water	0.9	0.15	Unaffected
36	25	3.5 grains in 1 ounce water	0.75	0.15	Unaffected
37	17	3.5 grains in ½ ounce mineral oil	1.5	0.2	Sickened but recovered
38	16	4.2 grains in 2 ounces water	0.45	0.25	Died of nicotine poisoning

Various Technics Employed

As no great efficacy resulted from the use of various drugs on ten dogs (28 to 37, table II), it was decided to try other technics. The results of the experimental treatment by the rubber colon-tube and other methods, using various drugs and combinations of drugs, are given in table IV. The dogs recorded in tables I and II have been given identical numbers in table IV. Table II represents the first treatment on dogs 28 to 37, as shown in table IV. No toxic effects from the use of any vermicide were noted except in the cases of dogs 37 and 38, as reported in table III. The other technics are here described.

Table IV—*Data on 63 treatments of 27 dogs.**

Dog	Treatment	Drugs	Method*	Whipworms Passed	Later Findings
28	1st	Oil of chenopodium, arecolin, croton oil, santonin, oil of turpentine and tetrachlorethylene in mineral oil	C	134	Ova
	2nd	Same	C	0	Ova
	3rd	Nicotine in water	C	43	Negative
29	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	13	Negative
30	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	44	Ova
	3rd	Tetrachlorethylene in water	C	16	Negative
31	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	3	Ova
	3rd	Tetrachlorethylene in water	C	0	Ova
32	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	18	Negative
33	1st	Same as dog 1—1st	C	11	Whipworms on autopsy
35	1st	Nicotine in water	C	26	Negative
34	1st	Oil of chenopodium in mineral oil	C	26	Ova
	2nd	Nicotine in mineral oil	C	11	Ova
	3rd	Nicotine in mineral oil	C	104	Ova
	4th	Nicotine in water	C	1	Negative
36	1st	Nicotine in water	C	0	Ova

*Table II presents the first treatment of dogs 1 to 10, inclusive, in this table.

TABLE IV—*Data on 63 treatments of 27 dogs—continued.*

DOG	TREATMENT	DRUGS	METHOD	WHIPWORMS PASSED	LATER FINDINGS
37	1st	Nicotine in mineral oil	C	10	Whipworms on autopsy
38	1st	5 gallons physiologic saline at 108° F.	C	0	Ova
	2nd	Vaseline at 120° F.	C	6	Ova
	3rd	Nicotine in water	GCB	530	Ova
	4th	Nicotine in 1% soap solution	GCB	51	Ova
	5th	Nicotine in water	G	2	Ova
	6th	Nicotine in water	G	Died of nicotine poisoning	In cecum many dead, 18 live worms
39	1st	Santonin in castor oil	C	10	No further record
40	1st	5 pints 1% vinegar in 1% soap solution	C	0	Ova
	2nd	Nicotine in water	C	0	Ova
41	1st	Nicotine in water	C	0	Ova
	2nd	Nicotine in water	G	18	Ova
42	1st	Nicotine in 1% soap solution	C	22	Negative
43	1st	Tetrachlorethylene in vaseline at 118° F.	C	34	Ova
	2nd	Vaseline at 118° F.	C	0	Ova
	3rd	Nicotine in water	GCB	75	Ova
	4th	Nicotine in 1% soap solution	GC	46	Negative
44	1st	Nicotine in vaseline at 108° F.	C	0	Ova
	2nd	Vaseline at 115° F.	C	0	Ova
	3rd	Nicotine in water	GCB	52	Ova
	4th	Nicotine in 1% soap solution	GC	1	Negative
45	1st	Hexylresorcinol S. T. 37	GC	0	Ova
	2nd	Nicotine in 1% soap solution	G	0	Ova
	3rd	Nicotine in 1% soap solution	G	50	Ova
46	1st	Nicotine in 1% soap solution	G	22	Ova
	2nd	Hexylresorcinol S. T. 37	GC	0	Ova
	3rd	Nicotine in water	C	0	Ova
	4th	Nicotine in water	G	107	Ova
	5th	Nicotine in water	GC	Stool lost	No further record

Various Technics Employed

As no great efficacy resulted from the use of various drugs on ten dogs (28 to 37, table II), it was decided to try other technics. The results of the experimental treatment by the rubber colon-tube and other methods, using various drugs and combinations of drugs, are given in table IV. The dogs recorded in tables I and II have been given identical numbers in table IV. Table II represents the first treatment on dogs 28 to 37, as shown in table IV. No toxic effects from the use of any vermicide were noted except in the cases of dogs 37 and 38, as reported in table III. The other technics are here described.

Table IV—*Data on 63 treatments of 27 dogs.**

Dog	Treatment	Drugs	Method*	Whipworms Passed	Later Findings
28	1st	Oil of chenopodium, arecolin, croton oil, santonin, oil of turpentine and tetrachlorethylene in mineral oil	C	134	Ova
	2nd	Same	C	0	Ova
	3rd	Nicotine in water	C	43	Negative
29	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	13	Negative
30	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	44	Ova
	3rd	Tetrachlorethylene in water	C	16	Negative
31	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	3	Ova
	3rd	Tetrachlorethylene in water	C	0	Ova
32	1st	Same as dog 1—1st	C	0	Ova
	2nd	Tetrachlorethylene in water	C	18	Negative
33	1st	Same as dog 1—1st	C	11	Whipworms on autopsy
35	1st	Nicotine in water	C	26	Negative
34	1st	Oil of chenopodium in mineral oil	C	26	Ova
	2nd	Nicotine in mineral oil	C	11	Ova
	3rd	Nicotine in mineral oil	C	104	Ova
	4th	Nicotine in water	C	1	Negative
36	1st	Nicotine in water	C	0	Ova

*Table II presents the first treatment of dogs 1 to 10, inclusive, in this table.

TABLE IV—*Data on 63 treatments of 27 dogs—continued.*

Dog	Treatment	Drugs	Method	Whipworms Passed	Later Findings
37	1st	Nicotine in mineral oil	C	10	Whipworms on autopsy
38	1st	5 gallons physiologic saline at 108° F.	C	0	Ova
	2nd	Vaseline at 120° F.	C	6	Ova
	3rd	Nicotine in water	GCB	530	Ova
	4th	Nicotine in 1% soap solution	GCB	51	Ova
	5th	Nicotine in water	G	2	Ova
	6th	Nicotine in water	G	Died of nicotine poisoning	In cecum many dead, 18 live worms
39	1st	Santonin in castor oil	C	10	No further record
40	1st	5 pints 1% vinegar in 1% soap solution	C	0	Ova
	2nd	Nicotine in water	C	0	Ova
41	1st	Nicotine in water	C	0	Ova
	2nd	Nicotine in water	G	18	Ova
42	1st	Nicotine in 1% soap solution	C	22	Negative
43	1st	Tetrachlorethylene in vaseline at 118° F.	C	34	Ova
	2nd	Vaseline at 118° F.	C	0	Ova
	3rd	Nicotine in water	GCB	75	Ova
	4th	Nicotine in 1% soap solution	GC	46	Negative
44	1st	Nicotine in vaseline at 108° F.	C	0	Ova
	2nd	Vaseline at 115° F.	C	0	Ova
	3rd	Nicotine in water	GCB	52	Ova
	4th	Nicotine in 1% soap solution	GC	1	Negative
45	1st	Hexylresorcinol S. T. 37	GC	0	Ova
	2nd	Nicotine in 1% soap solution	G	0	Ova
	3rd	Nicotine in 1% soap solution	G	50	Ova
46	1st	Nicotine in 1% soap solution	G	22	Ova
	2nd	Hexylresorcinol S. T. 37	GC	0	Ova
	3rd	Nicotine in water	C	0	Ova
	4th	Nicotine in water	G	107	Ova
	5th	Nicotine in water	GC	Stool lost	No further record

TABLE IV—*Data on 63 treatments of 27 dogs—concluded.*

DOG	TREATMENT	DRUGS	METHOD	WHIPWORMS PASSED	LATER FINDINGS
47	1st	Hexylresorcinol S. T. 37	GC	0	Ova
	2nd	Nicotine in water	C	71	Ova
	3rd	Nicotine in water	GC	15	Negative
48	1st	Hexylresorcinol S. T. 37	GC	0	Ova
	2nd	Nicotine in 1% soap solution	G	0	Ova
	3rd	Nicotine in water	G	103	Ova
	4th	Nicotine in water	G	2	Negative
49	1st	Nicotine in water	G	27	Ova
	2nd	Nicotine in water	GC	14	Negative
50	1st	Nicotine in 1% soap solution	G	14	Negative
51	1st	Nicotine in water	GC	4	Ova
52	1st	Nicotine in water	GC	75	Negative
53	1st	Nicotine in water	GC	46	Negative
54	1st	Nicotine in water	GC	27	Ova

Abbreviations used: C, rubber colon-tube alone; GCB, glass tube, rubber colon-tube and balloon; G, glass tube alone; GC, glass and rubber colon-tubes.

Negative was recorded only after several negative microscopical findings over several weeks.

Glass tube alone: The enematized and anesthetized dog was hung head downward. A straight glass tube (12 mm or approximately ½ inch outside diameter) was inserted the full length of the descending colon. Nicotine solution was poured into the tube, which then was withdrawn. Then the dog was laid on its right side and the front end slowly raised. It was hoped that gas bubbles would pass from the cecum into the colon. No increase in efficacy resulted from the use of this method (table V).

Balloon retraction: A toy balloon, fastened over the end of a No. 22 Davol tube, was passed through the inserted glass tube and advanced as far as possible. Next the balloon was inflated by mouth through the rubber tube and the rubber tube and attached inflated balloon retracted slightly. The balloon was deflated and the process repeated several times. A dog, autopsied at this point, showed the entire colon now in a straight line and wrinkled over the glass tube, much as a sleeve may be wrinkled up and shortened on an arm. The cecum was now close

TABLE V—*Tabulation of data in table IV.*

Drug	Rubber Colon-Tube		Glass Tube		Glass and Rubber Tubes and Balloon		Glass and Rubber Tubes	
	Result	Later Findings	Result	Later Findings	Result	Later Findings	Result	Later Findings
Mixture*	Passed worms 2 (28.6%) Passed no worms 5 (71.4%)	Negative 0 (0%) Ova or worms 7 (100%)			Passed worms 2 (100%)	Negative 0 (0%) Ova 2 (100%)		
Nicotine	Passed worms 8 (61.5%) Passed no worms 5 (38.5%)	Negative 4 (30.8%) Ova or worms 9 (69.2%)	Passed worms† 8 (66.7%) Passed no worms 4 (33.3%)	Negative 2 (16.7%) Ova or worms 10 (83.3%)	Passed worms 2 (100%)	Negative 0 (0%) Ova 2 (100%)	Passed worms 8 (100%)	Negative 6 (75%) Ova 2 (25%)
Tetrachlorethylene	Passed worms 6 (85.7%) Passed no worms 1 (14.3%)	Negative 3 (42.8%) Ova 4 (57.2%)						
Oil of chenopodium	Passed worms 1 (100%)	Negative 0 (0%) Ova 1 (100%)						

*See first treatment given to dog 28, table IV.
†See sixth treatment, dog 38, table IV. Live and dead worms found on necropsy.

Notes:
Eleven treatments are not considered because of:
(1) Impotent drugs (38-1, 38-2, 40-1, 43-2, 44-2, 45-1, 46-2, 47-1, 48-1).
(2) No further record (39-1, 46-5).

to the end of the glass tube. With the dog held hind end up, the rubber tube and attached balloon were withdrawn and the solution poured through the glass tube. The cecum filled completely. However, in practice, this method was not much more efficacious (table V).

Glass and rubber tubes: A No. 22 Davol tube was passed through the inserted glass tube until great resistance was met. The injection was now made through the rubber tube. Naturally, there was no bellying of the descending colon and less coiling than when the rubber tube alone was used. This method gave the best results of any used up to this time (table V).

Table V represents a tabulation of the data given in table IV. The ranking of the various technics according to their efficacy apparently was:

1. Glass and rubber tubes.
2. Glass and rubber tubes and balloon.
3. Glass tube.
4. Rubber tube.

A perusal of table V does not clearly indicate which drug is the best trichuricide. While all may be equally effective, there seems to be some superiority in nicotine.

True Intracecal Injections

A critical review of the work done up to this point and work on eight more experiment dogs and cadavers resulted in a method of passing the tip end of the rubber colon-tube into the cecum itself, often right to the tip end of the cecum. Fasting for twelve to 24 hours was practiced. Next came thorough enematization and then nembutal anesthesia. The dog was placed on its left side. A lubricated, straight, thick-walled glass tube (½ inch outside diameter), with fired ends, was passed into the colon as far as possible.

Work on cadavers, with the flank laid open as a flap, showed that the colon was now tensed with the transverse portion unrecognizable. The colon now consisted of a descending portion enveloping the glass tube, an acute angle at the anterior end of the glass tube, and an ascending portion. Tension on the glass tube was now slightly reduced. A lubricated No. 22 or No. 28 Davol tube now was passed forward through the glass tube. Work on cadavers showed that in short-bodied dogs the rubber tube often passed directly into the cecum. In long-bodied dogs, the tube tip reached the immediate region of the ceco-colic orifice. During its progression one or more coils often developed in the ascending colon and its enclosed rubber tube.

Under moderate nembutal anesthesia, the dog would moan and stretch when the tube tip reached the vicinity of the ceco-colic orifice. Considerable back pressure was evident. If the hand holding the tubes at the anus was removed, the glass and enclosed rubber tube would move back out of the anus, usually until the entire glass tube protruded. However, the tip end of the rubber tube, in the vicinity of the ceco-colic orifice, did not move in its relationship with the ceco-colic orifice. Rather, uncoiling occurred and the ascending colon shortened so that the cecum moved cranialward. Now the rubber tube was held stationary but the glass tube was advanced slowly until a mild resistance was met. This resistance was caused by the rightward bend of the rubber tube in the restored transverse colon. Pressure against this resistance resulted in pulling the rubber tube-end forward and away from the ceco-colic orifice, which was undesirable. The glass tube was now held stationary. Further advancing of the rubber tube alone resulted in its intracecal penetration.

The success of the entire technic depends on the development of the proper sense of touch. Resistance is met first as the tip end of the rubber tube leaves the anterior end of the glass tube. Sudden resistance followed by an easy advance means that the tube-end has turned on itself and is moving back toward the anus or that a sharp, S-shaped bend has occurred in the tube. A moderate, gradually increasing resistance characterizes the proper, gradual advance of the tube.

With the dog lying on the left side, and by wetting the right flank, to lay the hair, the tip end of the tube usually can be seen to cause an advancing bulge in the right flank as it approaches the cecum. When the tube-end is stopped in the cecum, extreme resistance is felt, with the dog moaning and stretching. Palpation through the abdominal walls is informative. If the rubber tube doubles on itself, the glass tube may be felt through the abdominal wall with the rubber tube closely applied to it. It is impossible to palpate the rubber tube at its most anterior part, but it may be felt in the right flank. When the operator believes the tube has entered the cecum, an assistant holds the glass and rubber tubes stationary.

A distinctive feature is the last curve or hook of the tip end of the rubber tube as felt through the belly wall. The extreme tip end of the tube may be bent sharply enough to obliterate its lumen. This characteristic bending is caused by the peculiar conformation of the cecum which causes the tube-end to bend sharply. Experimental attempts to puncture the cecum were un-

successful. Often, under extreme pushing, the tube-tip would bend in the cecum and double back toward the colon.

When the operator is satisfied that the tube-end lies in the cecum, the hold on the tubes at the anus is slightly relaxed. This restores the actual lumen in the tube-end. Injections now are made directly into the cecum through the rubber tube. Standard nicotine solution (1 Lilly Pulvule No. 142 in 1 ounce of water; dose, ½ ounce to 2 ounces, according to size) was used in thus treating 24 infested dogs (table VI). The No. 28 tube was more satisfactory. The No. 22 tube was used in small dogs, but gave more trouble by doubling on itself.

TABLE VI—*Data on true intracecal injections of standard nicotine solution.*

DOGS	TREATMENTS	RESULTS	LATER FINDINGS
63	1	Whipworms passed	No further record
64-81	1	Whipworms passed	Negative
82-85	2	Whipworms passed after each treatment	Negative
86	3	Whipworms passed after each treatment	Negative

Beneficial results followed each treatment. No harm to any dog treated was noted. Often, when the tubes were withdrawn, whipworms would be found adhering to the tip end of the rubber tube. Whipworms were passed after each treatment, but four dogs required two treatments each, and one dog required three treatments to become negative (table VI). Eighteen dogs (64 to 81) of the 24 shown in table VI became negative after one treatment. This is an efficacy of 75 per cent.

INTRACECAL EXPULSION OF NICOTINE CAPSULES

Further experimental work on five dogs (87 to 91, table VII) showed that it is possible to expel a nicotine capsule directly into the cecum. Three dogs (92, 93 and 94) are included in table VII because the cecum of each was found free of whipworms three days after the capsule expulsion. No indication of irritation was noted in the cecum or colon of any of the eight necropsied dogs shown in table VII.

First, the lubricated No. 28 Davol tube is passed through the glass tube until its tip end protrudes. A pulvule is covered with petrolatum and pushed into the tip end of the rubber tube. (With a new tube it is necessary to stretch the lumen of the tip by the insertion of a large probe.) The glass tube is lubricated and

the entire apparatus is inserted into the colon. The technic now follows exactly the one described under "True Intracecal Injections."

TABLE VII—*Autopsy results following intracecal nicotine capsule expulsion.*

DOGS	FECAL	WHEN AUTOPSIED	RESULTS*
87-88	Negative	Immediately	Capsule broken open in expulsion. Contents in cecum
89-90	Negative	Immediately	Unbroken capsule found in cecum
91	Whipworm ova	1 hour	Nicotine seen in cecum. All whipworms dead but fast to mucosa
92-94	Whipworm ova	72 hours	Passed worms within 48 hours. Cecum empty at 72 hours

*No indication of irritation was noted in the cecum or colon of any of the dogs.

When the operator is sure that the tube-tip is in the cecum, a metal syringe is inserted in the exposed end of the rubber tube and water injected with considerable pressure. The capsule pops out with quite a loud report and is deposited directly in the cecum, where it dissolves. With smaller dogs, half the content of one pulvule is discarded. The liberated nicotine kills the whipworms which are expelled in twelve to 96 hours. In two of 192 dogs (table VIII) so treated, the capsule was expelled through the anus immediately after intracecal expulsion, but was retained on immediate retreatment. In eleven treatments (dogs 277 to 286, table VIII),* where ova were noted after treatment, though three treatments (dogs 277, 278 and 279) resulted in passage of whipworms, it is believed that too soft a tube was used.

Washing the rubber tube with boiling water, after it is used each time, as well as the use of petrolatum as a lubricant, causes the tube to soften and swell. In such cases, characteristic bending of the tube-tip was not felt, and it is believed that the pulvule was not deposited in the cecum. The use of a new tube immediately resulted in 100 per cent efficiency. Of the entire group of 192 dogs shown in table VIII, 179 (93.2 per cent) had repeated negative fecals after one treatment.

In our current work, fasting has been reduced to an eight-hour period. The belly is massaged during enematization to soften any hard masses in the colon or cecum. Enematization is continued until only clear water returns from the colon. Anesthesia is considered essential to success.

*Dog 280 received two treatments.

A colleague, Dr. Alfred Kissileff, of Flourtown, Pa., uses this technic but has substituted a section of an equine stomach-tube for the glass tube. This is softer but still rigid enough to prevent coiling.

In three instances, the rubber colon-tube, instead of entering the cecum, penetrated through the ileo-colic valve and entered

TABLE VIII—*Data on intracecal expulsion of nicotine capsules.*

DOGS	TREATMENTS	WHIPWORMS PASSED	LATER FINDINGS
179 (95-273)	1	+	Negative
1 (274)	1	+	Condition improved but no further fecal record
2 (275-276)	1	Sent home	No further record
3 (277-279)	1 2	+ +	Ova Negative
1 (280)	1 2	— —	Ova Ova
6 (281-286)	1 2	— +	Ova Negative

the ileum. This occurrence is easily recognizable, because instead of turning into the cecum, the tube can be palpated easily as it passes caudalward in the right flank as far as the pelvis. The tube then will turn cranialward and coil through the small intestine. In one of these three instances, a nicotine capsule was expelled well into the ileum. Shortly afterward, although well under nembutal anesthesia, the dog passed many dead tapeworms. The following day he passed whipworms. Probably the nicotine was in sufficient concentration to kill the whipworms as it passed through the ileo-colic and ceco-colic openings.

Early in the experimental work, it was decided to test the absorptive powers of the cecum. A cecotomy was performed and a 00 capsule filled with methylene blue powder was inserted into the tip end of the cecum. Urine and feces become blue 36 hours later, possibly indicating poor absorption from the cecum, with absorption occurring in the colon. Therefore, it would appear that there is less danger of toxic reaction with the intracecal capsule expulsion method than with the intracecal injection of

nicotine solution. Liquids pass out of the cecum shortly after injection. The nicotine in capsule form would require many hours to leave the cecum.

SUMMARY

The life history of *Trichuris depressiusculus*, the dog whipworm, is given. Infestation is by ingestion of embryonated ova. No intermediate host or free-living stage occurs. The larvae enter the cecum and when sexually mature extrude ova which reach the outside world in the feces of the host.

The symptoms produced by whipworm infestations are principally eczema, lassitude and occasional diarrhea. Diagnosis is made by finding the characteristic ova in the feces.

Whipworms produce only occasional gross pathology in the cecum. Possibly the symptoms are due to enteric absorption, by the host dog, of a toxic whipworm excretion.

Treatments commonly in use are oral and intracolonic administration of various vermicides. Both methods usually produce disappointing results. Cecectomy results in complete and permanent elimination of the parasite, but clients do not favor this operation.

From the anatomical standpoint, the colonic flexures and the flexibility of the colon interfere with the passage of a rubber colon-tube to the cecum.

Experimental rubber-colon-tube passage in our hands indicated that, with proper attention to certain details, it was probable that the cecum could be filled with a vermicidal liquid in the majority of cases. These details were moderate fasting, thorough enematization, the proper type of tube (No. 22 to 28 Davol) and anesthesia. However, experimental intracolonic injections of vermicides produced no brilliant results.

Trichuricidical tests on living whipworms attached to excised ceca established nicotine as the drug of choice.

Nicotine toxicity tests established a safe but effective dosage of 0.7 grain of nicotine in solution for the average dog. A dose of 3.5 grains in solution caused a 17-pound dog to sicken but he recovered, whereas 4.2 grains in solution killed a 16-pound dog.

Injecting nicotine solution by gravity, through a straight glass tube inserted the full length of the colon, was not very effective. A combination of glass and rubber tubes, with a balloon over the end of the rubber tube, was used to retract the cecum, but this method was not very effective either. The best results were secured by inserting a glass tube the entire length of the descending colon and then passing a rubber colon-tube through the lumen of the glass tube. This technic resulted in less coiling. The

vis a tergo was better transmitted into an advancement of the tube-end.

In tabular form data are presented to show the best results from the use of the glass and rubber tubes and nicotine solution. This combination resulted in 75 per cent efficacy in one treatment on eight dogs. The treatment was considered efficacious if the dog passed whipworms and showed repeated negative fecals for several weeks. True intracecal penetration of the tube-tip was proven practical. The technic has been described. An efficacy of 75 per cent resulted from intracecal injections of nicotine solution.

A method has been described for the intracecal expulsion of nicotine in capsules. Up to date, 179 (93.2 per cent) of 192 dogs thus treated have passed whipworms and have remained negative after one treatment. The capsules used are known as Lilly Pulvules No. 142.

Conclusion

Although much of the work described in this paper lacks exactness, it is believed that the comparative values of various technics and drugs are shown well enough for practical purposes. The work was not carefully planned in advance but the author has numbered and grouped the treated dogs so as to give a fairly clear and chronological picture of the development of the studies. The author is satisfied that the intracecal expulsion of capsules containing nicotine, or other recognized vermicides, in proper dosage, is safe, practical and efficacious.

Training of the sense of touch and practice on fresh cadavers and experiment dogs is imperative for success. Improvement, often startling, has been noted in every dog successfully treated for whipworm infestation. A few dogs successfully treated last winter came back this summer with a recurrence of symptoms and whipworm ova again were found. This showed reinfestation, probably from ova-infested exercise grounds. The writer advised cecectomy, but in each instance the owner insisted on some other treatment. It is our opinion that where heavily infested exercise yards exist, cecectomy is advisable unless the yard infestation can be remedied. Cecectomy is not necessary in house and apartment pets kept off infested grounds.

Acknowledgments

Thanks are due Dr. W. H. Wright, Zoölogical Division, U. S. Bureau of Animal Industry, and Dr. W. J. Lentz, School of Veterinary Medicine, University of Pennsylvania, for constructive

criticism. Thanks are due Messrs. M. Gradess and L. Sherman, veterinary students at the University of Pennsylvania, for help in this work.

REFERENCES

[1]Wright, W. H.: The incidence of internal parasites in dogs at Washington, D. C. Jour. A. V. M. A., lxxvi (1930), n.s. 29 (6), pp. 794-803.
[2]Underwood, P. C., Wright, W. H., and Bozicevich, J.: Critical experiments with the intracecal method of injection for the treatment of whipworms in dogs. No. Amer. Vet., xii (1931), pp. 41-47.
[3]Steinbach, F. G.: Effective whipworm treatment on dogs. Vet. Med., xxv (1930), p. 543.
[4]Steinbach, F. G.: Additional notes on whipworm elimination. Vet. Med., xxvii (1932), pp. 480-481.
[5]Steinbach, F. G.: Personal communication, 1934.

More Purebred Animals Being Imported

During the year ended June 30, 1935, according to a report of the U. S. Bureau of Animal Industry, there were 10,836 purebred animals imported into the United States for breeding purposes. This number was about one-third greater than for either the year 1933 or 1934. The figures for recent years are as follows:

1930	12,843
1931	7,818
1932	10,647
1933	6,939
1934	7,411

It will be noted that the number for 1935 was greater than for any year since 1930. The tariff act of that year provided for the free entry of purebred animals by American citizens for improving herds and flocks. The 10,836 animals imported during 1935 were divided as follows:

Cattle	8,663
Horses	274
Sheep	1,036
Swine	12
Dogs	839
Cats	12

Nine breeds of cattle were included in the 1935 importations, which were distributed as follows:

Aberdeen Angus	33
Ayrshire	2,119
Brown Swiss	2
French Canadian	36
Guernsey	26
Hereford	220
Holstein-Friesian	5,531
Jersey	600
Shorthorn	96

The importations of horses were distributed among nine breeds, with Belgians predominating. Nine breeds of sheep were among those entering the country, with Southdowns and Suffolks accounting for 861 of the 1,036 sheep imported.

THE DIAGNOSIS OF FILARIASIS IN THE DOG*

By E. L. STUBBS *and* I. LIVE
School of Veterinary Medicine, University of Pennsylvania
Philadelphia, Pa.

The diagnosis of filariasis in the dog is usually made by demonstrating moving larvae in freshly drawn blood examined immediately. We have found that it is not necessary to examine the blood immediately. We now prefer that a blood sample be drawn and allowed to clot, and the examination made of the serum. The microfilariae can be detected moving about in the serum and they appear to be more active than in whole blood, probably because they do not have to overcome the resistance of the blood cells that are present in whole blood. The microfilariae can also be seen in the blood serum even when motionless and presumably dead. It appears that the microfilariae are more numerous in the serum because they wriggle out from the clot as it forms and are found in the serum. They may be more numerous because they are concentrated in a smaller volume when in the serum than when in the whole blood, or the serum may form less impediment to their movement than when they must overcome the resistance of the blood cells in the whole blood.

Routine postmortem examinations of dogs occasionally show dirofilariae in the heart of a dog in which the disease was not suspected. The finding of filariae at postmortem raised the question as to whether living larvae could be demonstrated in dogs coming to postmortem and the conditions under which blood from living dogs could be examined for filariae. We had no difficulty in demonstrating living larvae in dogs coming to postmortem even in dogs dead longer than 24 hours. We also found that the microfilariae could be even more easily demonstrated in serum from the blood that had clotted, and that it was not necessary to examine blood immediately after being drawn. This has led to the examination of many blood samples for the presence of

*Received for publication, November 11, 1935.

microfilariae and these parasites have been found in a wide variety of breeds and conditions of dogs. Formerly it was the practice to examine the blood for microfilariae only of dogs from the South, hunting dogs, or dogs that exhibited some heart or circulatory disturbance. Now it is our practice to examine the blood of many other dogs, particularly those cases undiagnosed or of an obscure nature.

This examination of blood for filariasis may be made immediately after it is obtained or at a later time. When the whole blood is to be examined it must be done immediately before the blood clots, or an anticoagulant may be used. For such an examination we prefer a blood sample consisting of 2 or 3 cc of blood collected with a sterile syringe from the recurrent tarsal vein and placed in a sterile test-tube or vial. The blood is allowed to clot and the serum to separate. A drop of serum is removed by a medicine-dropper or a glass rod and placed on a slide. A cover-slip is dropped over the serum. The cover-slip area is covered systematically with the low power and the microfilariae can be plainly seen. When the serum is not examined for some time and there has been a chance for settling, the microfilariae may settle to the bottom. The supernatant fluid may be poured off and the lower layers examined, or the vial may be inverted a few times to make an even mixture of the parasites. The microfilariae are plainly visible in the serum even though inactive or dead, while in whole blood the erythrocytes obstruct the view and motion of all except the very vigorous ones. It is possible that during the clotting the microfilariae wriggle enough to get out of the clot and are found in the serum.

We have also tried the concentration method, as recommended in Kolmer and Boerner's "Approved Laboratory Technic," of mixing blood with 2 per cent acetic acid, but prefer to let the blood clot and examine the serum.

Routine examination of blood samples for microfilariae presented the opportunity for making studies on the viability of microfilariae outside the animal body under various conditions. The studies were made on blood samples routinely collected and submitted for examination. Some were blood samples collected and allowed to clot, and others were collected with the addition of an anticoagulant. Some were placed in the ice-box and others kept at room temperature. Daily examinations were then made for microfilariae. Only blood samples that showed numerous active microfilariae were selected for study. One group of blood samples were selected from those that had been collected without the use of an anticoagulant. This group was kept

in an electric refrigerator that had a temperature of about 8°C. Under such conditions, when examined immediately after removal from the ice-chest, the microfilariae moved sluggishly, but when warmed to 37°C. they again became active. Blood samples in this group examined daily showed living filariae as determined by active movement up to the 15th day. No active microfilariae were found after the 15th day. Blood samples collected with an anticoagulant and kept in the ice-chest showed no active microfilariae after the ninth day.

Blood samples collected without an anticoagulant and kept at room temperature were hemolyzed after two days and then showed no active microfilariae.

Blood samples collected with an anticoagulant and kept at room temperature were hemolyzed after one day and no living parasites could be detected.

Our studies indicate that microfilariae are present in many cases not suspected. The practicability of collecting a blood sample to be examined later should widen the usefulness of such a method of examination in small-animal practice.

ADDITIONAL AMERICAN RECORDS OF DIOCTOPHYME RENALE FROM THE DOG*

By WILLARD H. WRIGHT, *Washington, D. C.*
Zoölogical Division, Bureau of Animal Industry
U. S. Department of Agriculture

Recently Underwood and Wright[1] attempted to summarize the published American records of the occurrence of the giant kidney worm, *Dioctophyme renale*, in the dog. Since the publication of that note, additional cases have come to the attention of the writer. One of these cases was referred to him by Dr. D. E. Buckingham, Washington, D. C., to whom the writer is indebted for his interest in the matter. This case involved a 3-year old female setter, the property of John E. McPherson. On the day of its death, this dog received a workout in the field but as it became very tired after a run of an hour, the owner returned home with it. At 9:00 o'clock that evening, the dog endeavored to vomit and became very dyspneic. The owner placed the dog in his automobile with the intention of taking it to Dr. Buckingham's hospital but the dog died en route. Necropsy performed by Dr. Buckingham revealed two large female *D. renale* in the

*Received for publication, November 18, 1935.

abdominal cavity. These nematodes were 47.752 and 85.090 centimeters (18.75 and 33.5 inches) long, respectively. The abdominal cavity contained a large amount of sanguineous serous fluid, from which numerous ova of *D. renale* were recovered. There was present a marked chronic peritonitis with numerous fibrous attachments of the peritoneum to the liver and spleen. On two different occasions, the dog in question had spent some time in training near Sumter, South Carolina, where it is possible that the infestation was contracted.

Underwood and Wright summarized, among others, cases recorded by Riley.[2, 3] Among these cases, Riley[2] quotes Stiles to show that Welch and others had found *D. renale* in dogs at Baltimore, Md. Riley, in his summary, apparently counts this record as one case. A search of the literature has since revealed that Welch,[4] up to 1890, had encountered the parasite in dogs on three occasions. In addition to Welch's report, Crowe[5] published a record of one case of *D. renale* found in a dog at Baltimore.

The present report adds four additional cases to those previously definitely recorded.

REFERENCES

[1]Underwood, P. C., and Wright, W. H.: A report of the giant nematode, *Dioctophyme renale*, from a dog, with a summary of American records. Jour. A. V. M. A., lxxxv (1934), n. s. 38 (2), pp. 256-258.

[2]Riley, W. A.: The occurrence of the giant nematode, *Dioctophyme renale* (*Eustrongylus*) in the United States and Canada. Jour. A. V. M. A., xlix (1916), n. s. 2 (6), pp. 801-809.

[3]Riley, W. A.: Review of previous reports of *Dioctophyme renale*. Jour. Parasitol., xi (1925), 4, p. 229.

[4]Welch, W. H.: Report of remarks and exhibition of specimens of animal parasites, meeting Johns Hopkins Hospital Medical Society, March 17, 1890. Johns Hopkins Hospital Bul., 1 (1890), 6, pp. 72-73.

[5]Crowe, S. J.: The parasites of Baltimore dogs. Johns Hopkins Hospital Bul., 18 (1907), No. 201, pp. 464-467.

Texas Leads in Sheep Production

Texas has 6,625,000 of the estimated 42,985,000 sheep in the United States, according to the *Cattleman*. Other leading sheep-producing states are: California, 3,503,000; Montana, 3,350,000; Wyoming, 3,174,000; Oregon, 2,275,000, and New Mexico, 2,264,000.

Rochester to Be Host to A. A. A. S.

The 1936 summer meeting of the American Association for the Advancement of Science will be held in Rochester, N. Y., June 16-18. Immediately following the meeting, on June 19-20, the Society of Sigma Xi will celebrate the fiftieth anniversary of its founding. The celebration will be held at Cornell University, at Ithaca.

VETERINARY MILITARY HISTORY OF THE UNITED STATES, With a Brief Record of the Development of Veterinary Education, Practice, Organization and Legislation. Louis A. Merillat, Lt. Col., Vet.-Res.; Chief Veterinarian, First Army, American Expeditionary Forces, and Delwin M. Campbell, Lt. Col., Vet.-Res.; Editor, *Veterinary Medicine*. Sponsored by the American Veterinary Medical Association. 2 vol., 1172 pages, with 476 illustrations. Veterinary Magazine Corporation, Chicago, and Haver-Glover Laboratories, Kansas City, Mo., 1935. Price, $10.

Volume II is divided into three books, covering the period 1916-1935. It contains 552 pages and is as profusely illustrated as volume I. This part is devoted entirely to military history and adds justification to the title, which, after reading the first volume, we concluded did not adequately describe the subject covered.

The first book (VIII) of this volume is devoted to the activities of the Veterinary Corps of the American Expeditionary Forces in France during the World War. A perusal of this section will refresh one's knowledge of American participation in the great conflict.

Those who boast with pride of American horsemanship will be decidedly disillusioned when they read of the conditions that general ignorance and improper handling of animals brought on, even to the point of attracting international attention and the appointing of an international veterinary committee to correct the situation.

We are told of the handicaps and tribulations the newly created Veterinary Corps experienced. It was much like an orphan mothered unsympathetically by the Remount Division of the Quartermaster Corps assisted by the cavalry, before it ultimately found its natural mother, the Medical Corps, almost too late, but nevertheless in time to save its reputation.

The functioning of the veterinary service with the higher combat units (corps and army) is well described, although the subject is approached from an entirely different angle than volume I.

This phase may be termed the senior author's personal memoirs. Of necessity a record of one's personal experiences are bound to reflect the viewpoint and personal feelings of the writer. Particularly is this true when the event is recent and the memory of it fresh. However, it must be remembered that Dr. Merillat played a most important part in the drama portrayed. Also sufficient evidence is shown that the authors have made every effort to obtain as much material as possible, from other than personal sources, by the fact that complete chapters by Cotton, Marshall, Turner, Wright and others have been included. The criticism, if any, must be leveled against the members of our profession for failing to record in writing their experiences and failing to contribute important data to veterinary history. By neglecting to do so, they have withheld much of interest from their professional brothers.

A chapter entitled, "Veterinary Service with the Field Artillery of the American Expeditionary Forces," by Willard H. Wright gives first-hand experiences with the artillery and is very interesting. Another chapter on "Hospitalization of Animals" outlines the method of hospitalizing horses and mules and the difficulties met by the veterinary hospitals. It is amusing to the reviewer to note that the authors have chosen the veterinary hospital (Bourbonne-les-Bains) in which he served as an example to describe how terrible they were.

"Post Armistice Veterinary Activities," "Condensed History of the A. E. F." and "Official Documents of Special Veterinary Interest" are all chapters of interest and education and conclude this part. Some personal criticisms appearing throughout this book seem rather harsh to us, but again we must admire the frankness and boldness that has been in evidence throughout the entire work.

As we complete the story, as told by these authors, of the participation of the American veterinarians in the World War, we can not but be pleased with their intelligence and ability as a whole. The veterinarian had been held down by army authorities to the level of a civilian employé, without rank, until 1916. His capabilities had not been tried beyond that of a regimental officer. In the brief space of two years, as war progressed and during its aftermath, we find him figuratively growing, qualifying in turn, first as a division, then as a corps, and again as an army officer and acquitting himself with honors. It must be admitted that as chief of his branch, both at home and abroad, he did not triumph. At home he was not permitted that high administra-

tive office and abroad he was found wanting. We must conclude that another six months would have found him qualified in this respect, also. His development and the difficulties he overcame are well told.

Book IX, the first chapter of which describes the structure and activities of the Veterinary Corps from 1920 to 1935, amply justifies the conclusion we have expressed in the preceding paragraph. The many achievements of the Corps are enumerated in this book.

A short chapter on "Dogs in the Military Service" is exceedingly interesting. One on gas warfare is most instructive and the concluding one, "Rail Shipment of Animals in Peace and War," should be read by all men interested in animal shipment whether in or out of the profession.

Book X concludes this excellent work and consists of 164 pages of lists of Army veterinarians. They include: Veterinarians Who Served in the Army of the United States; Veterinarians in the Old Army, 1866-1916; Veterinary Officers of the World War, 1917-1919; Officers of the Veterinary Corps, Regular Army, 1935, and The Veterinary Reserve, 1935.

After reading volume II, which is entirely of a military nature, we feel better pleased regarding the title. We nevertheless feel that a better purpose would have been served, had two books been issued under two separate titles (Military and Civil), each elaborating on its respective subject. We must consider, however, that the American veterinarian is not yet history-minded and that the reception of this book is the proverbial algebraic quantity x. To say the least, a "noble experiment."

Military interest for the average veterinarian is confined to those, who, for the most part, saw military service in the world turmoil, and those who are at the present time in the regular military establishment (only a handful). By including the civil aspect, the writers have, by interesting the entire profession, demonstrated their business sagacity. If, however, the proper interest is shown by veterinarians, we make bold to predict (without authority or permission), that the authors, than whom there are none better, or even nearly so well qualified, may eventually be persuaded to publish another book dealing solely with the civil history of our profession.

Until this comes to pass, we must rate "Veterinary Military History" as the best work of its kind in American veterinary literature.

J. M. A.

SEDGWICK'S PRINCIPLES OF SANITARY SCIENCE AND PUBLIC HEALTH. Samuel C. Prescott and Murray P. Horwood. 654 pages. Macmillan Co., New York, 1935. Price, $4.25.

Although based upon the book, "The Principles of Sanitary Science and Public Health," written by Professor Sedgwick in 1901, this edition has been completely rewritten and enlarged to embrace all the modern practices in public health.

The authors apologize for the absence of the pleasing style of writing found in the 1901 edition, but it would be difficult to criticize the simple, direct, and intensely interesting style presented in this edition. Examples chosen to illustrate are especially noteworthy. The authors' years of association with Professor Sedgwick were not spent in vain.

Every chapter in the book would be of interest to the veterinarian, but space does not permit its description. The following chapters are of particular interest and have direct bearing on the field of veterinary hygiene: Health, Old Age and Disease; The Etiology or the Causes of Disease; Ancient and Modern Theories; The Rise and Influence of Bacteriology; Sanitary Aspects of the Struggle for Existence: Factors Affecting Survival; Infection and Contagion: The Paths and Portals by Which They Enter the Body; Dirt, Dust, Air and Disease; The Living Earth; Milk Supplies and the Public Health (this chapter embraces 47 pages, covering all phases of the nutritional values of milk, the dangers found in milk and the methods by which safe milk may be produced); Certain Uncooked Foods (Meats, Oysters, Fruits, Vegetables, etc.) as Vehicles of Disease; The Prevention and Inhibition of Infection, Decomposition and Decay; The Destruction or Removal of Infection; Disinfection and Disinfectants; Rats and the Public Health; Flies and the Public Health; Air in Relation to Health and Comfort; Carbon Monoxide Poisoning; The Relationship of Housing to Health; Public Health Aspects of Tuberculosis; Organization of Public Health Administration in the United States. This chapter is a concise yet complete description of the organization of health units nationally, in the state, city and small community.

The book contains some inaccuracies in giving credit for the discovery of certain microörganisms but these are unimportant when considered with the many worthwhile features. It would seem that some space should have been allotted for the description of our federal meat inspection system. The chapter on uncooked foods mentions trichinosis, but nothing about tapeworm infestation due to beef and pork cysts. Salmonella infections

which may arise from uncooked meat are not mentioned. From the viewpoint of the veterinary sanitarian this is the weakest chapter in the book.

The book is highly recommended to those interested or working in the field of public health.

I. A. M.

VETERINÄRHYGIENE MIT ANLEITUNG FÜR DIE HYGIENISCHEN UEBUNGEN LEITFADEN FÜR BEAMTETE UND PRAKTISCHE TIERÄRZTE, SOWIE FÜR STUDIERENDE DER VETERINÄRMEDIZIN (Veterinary Hygiene with Directions for Carrying out Hygienic Work Prepared for Official and Practicing Veterinarians as well as for Students of Veterinary Medicine). Hermann Miessner, Professor of Hygiene, Infectious Diseases and Sanitary Science, and Director of the Institute of Hygiene at the Veterinary College at Hannover. Gerhard Schoop, Assistant Professor of Veterinary Bacteriology and Hygiene and Assistant in the Institute of Hygiene at the Veterinary College at Hannover. 171 pages, with 87 illustrations. Verlag von M. & H. Schaper, Hannover, 1935. Paper, M10; cloth, M11.50.

There is a lot of information contained in these 171 pages. There is not a book in English that is of exactly this character. This publication is not intended in any sense as a reference book except as a reference work may be used as a guide. References are largely omitted. It does, however, contain many interesting and instructive facts. It deals with the air and its composition, its contamination, and its significance for animal health. Soil is discussed under content such as porosity, type, appearance, germ content, temperature, and a little of its chemistry.

Water comes in for its share of discussion. We were particularly interested in the type of purification filters described. They do not in any sense compare to the large filter beds used by certain of our American municipalities. Stable construction, hygiene and ventilation receive due attention. Here, too, some of the methods of ventilation would not work any too well, at least not in the climate of our Northwest. Barn construction described varies considerably from the usual type in this country. External and internal parasites are briefly discussed. Disinfection and disinfectants do not, in the opinion of the reviewer, receive sufficient attention.

A discussion of feed and its relation to veterinary hygiene is also very brief. We cannot agree with some of the statements made in regard to the effect of molds, although the authors safe-

guard themselves to a great extent. Those to whom the language is not a barrier, and who wish a brief, concise statement of facts relating to veterinary hygiene and sanitation, would do well to procure this book.

C. P. F.

Pigeons Replace Telephones in Japan

Carrier pigeons still have plenty to do in Japan, where telephones are comparatively scarce. They are used frequently by Japanese newspaper reporters, who take them along when reporting news from out-of-the-way places. A speedy bird, by getting home a few minutes ahead of that of a rival newspaper, can often give its owners a "scoop."

Equine Influenza in London

More than 500 cases of equine influenza were reported in North London during the few weeks preceding August 17, 1935, according to the *Veterinary Record*. At the same time, a severe epizoötic of "gastric influenza" among cats, with a mortality of 80 per cent, was also reported.

England Forgets

England's Slaughter of Animals Act, passed in 1933, came into force January 1, 1934. Through some oversight, however, the veterinary officer of health and the veterinary surgeon are not mentioned in any part of the Act. The only officials specified are the Medical Officer of Health and the Sanitary Inspector.

Editorializing on the subject, the *Veterinary Record* says:

> Is it not curious that the one man who, by his scientific training, knows more about the antemortem and postmortem examination of the food animals—*viz.*, the qualified veterinary surgeon—is never even mentioned throughout the clauses of a humane Slaughter of Animals Act?
>
> Truly we English are a curious anomaly in our methods! We build veterinary training schools, and establish courses of study for a minimum of five years, which include the diseases of meat, as well as those of the animal from which that edible meat is obtained; and then we allow the final and authoritative opinions on each to be given by a doctor of human diseases and a sanitary inspector.

Hate and mistrust are the children of blindness.—WILLIAM WATSON.

THE PATHOLOGY OF RICKETS IN DAIRY CALVES. H. Ernest Bechtel, E. T. Hallman and C. F. Huffman. Abst. Jour. Dairy Sci., xviii (1935), 7, p. 432.

This study was based on five normal and 11 rachitic grade Holstein calves, varying in age from 150 to 520 days, while the duration of the disease varied from 38 to 212 days. Low vitamin-D rickets in dairy calves was characterized principally by changes in the bones. These changes were always preceded by decreased concentrations of calcium and/or inorganic phosphorus in the blood plasma. The costo-chondral junction at the ventral end of the rib was selected as the best index to rachitic changes in the skeleton. Histological changes were confined largely to a relatively small portion of the bone at the costo-chondral junction. Retarded provisional calcification of the cartilage matrix appeared to be the fundamental change in rickets. The most conspicuous changes were the irregular removal of cartilage by the embryonic marrow and the accumulation of excess osteoid tissue. Growth was an important modifying factor in rickets. More severe rickets was associated with more rapid growth.

BLINDNESS IN CATTLE OF NUTRITIONAL ORIGIN ASSOCIATED WITH CONSTRICTION OF THE OPTIC NERVE. L. A. Moore, C. F. Huffman and C. W. Duncan. Jour. Dairy Sci., xviii (1935), 7, p. 435.

Thirty cases of blindness have occurred which are different from the true vitamin-A type of blindness. The blindness is observed in calves following birth and in young growing dairy animals when a ration containing poor quality roughage has been fed. It is frequently associated with paralysis, weakness, spasms and poor reproduction or denoted by premature births and retained placenta. The blindness is due to atrophy of the optic nerve where it passes through the optic foramen, apparently due to pressure atrophy caused by improper development of the foramen. Corn silage, timothy hay and cod-liver oil contain the factor or factors necessary to prevent this type of

blindness. The evidence indicates that rations low in calcium and vitamin D are not directly responsible for this type of blindness. Six calves fed 10,000 units of vitamin A in the form of "caritol" developed blindness.

A Comparison of Methods of Detecting Streptococci in Freshly Drawn Milk Samples. Wayne N. Plastridge, E. O. Anderson and Francis J. Weirether. Jour. Dairy Sci., xviii (1935), 9, p. 583.

Microscopic examination of films prepared from incubated milk revealed the presence of the causative organism of chronic streptococcal bovine mastitis in a larger number of instances than (1) direct microscopic examination of films prepared from whole milk or sediment and (2) blood-agar plates inoculated with a 4-mm loopful of whole milk or a 4-mm loopful of sediment or 1 cc of a 1:10 dilution of the sample. In the absence of other laboratory evidence of mastitis, the finding of streptococci in incubated samples should not be taken as conclusive evidence of infection with the organism which is commonly responsible for chronic infectious bovine mastitis. The significance of the finding of streptococci in such instances can be determined only by isolation and identification of the streptococci found in the sample.

Some Observations on the Germicidal Efficiency of Chloramine-T and Calcium Hypochlorite. David B. Charlton and Max Levine. Jour. Bact., xxx (1935), 2, p. 163.

A study was made of the effect of temperature, concentration and reaction on the germicidal efficiency of chloramine-T and calcium hypochlorite on the spores of *B. metiens* (nov. sp.). For a given concentration of available chlorine as chloramine-T a rise of 10° C. resulted in a decrease in the killing time of about 82 per cent if the initial reaction was pH 6.0 and approximately 71.5 per cent at pH 8.7. Doubling the disinfectant concentration at a given reaction and constant temperature reduced the killing time about 40 to 60 per cent. The pH range studied was from 6.0 to 8.8. Increasing the acidity markedly reduced the killing time. Experiments with hypochlorite gave similar results. Available chlorine was not found to be a direct measure of the germicidal efficiency of the calcium hypochlorite studies. A solution containing 1,000 p.p.m. available chlorine was only slightly more germicidal than the same solution diluted with distilled water to 100 p.p.m. (the reaction was changed from pH 11.3 to 10.4

by dilution) and very much less efficient than 20 p.p.m. at reaction pH 8.3.

A Study of the Corynebacteria Associated with Diseases of Domestic Animals. I. A. Merchant. Jour. Bact., xxx (1935), 1, p. 95.

Corynebacterium pyogenes, C. pseudotuberculosis, C. renalis and *C. equi* are four species of Corynebacteria associated with diseases of domestic animals. *C. pseudotuberculosis* and *C. renalis* are the only two of the four to show a marked serological relationship. Orange-colored variants were isolated from three cultures; the variants resembled the parent cultures in morphology but were smooth and pigmented and failed to ferment carbohydrates, whereas the parent cultures were usually rough and cream-colored and demonstrated some fermenting ability. The morphological characteristics of the four species of Corynebacteria are described.

The Biological Significance of Copper and Its Relation to Iron Metabolism. C. A. Elvehjem. Physiol. Rev., xv (1935), 3, p. 471.

The amount of copper in all living matter varies greatly in different organisms as well as in different tissues of the same organism. Copper is necessary as a supplement to iron for hemoglobin formation in red-blooded animals. Milk is low in copper as well as iron. Increased requirements may be supplied by simple inorganic copper salts together with iron salts. Copper is essential for plant growth but its function other than its association with chlorophyll formation is unknown. It is also essential for yeasts and other microörganisms. In the animal body it is concerned with the transformation of ingested iron into hemoglobin. A complete understanding of the action of copper will bring us nearer an understanding of the fundamental processes of living matter.

Fluid Transport Movement (Rheokinetic) Through the Small Intestines. H. E. Never. Physiol. Abst., xx (1935), 3, p. 218.

The passage of fluid through the small intestine of the guinea pig was investigated *in vitro*. The rheokinetic movement is a specific activity of the small intestine, independent of peristalsis and segmentation. It consists of rhythmic contractions of the

longitudinal musculature with tonic contraction of the circular musculature. If fluid is passed through the intestine in an antiperistaltic direction, no intestinal movement against the stream are elicited. The passage of isotonic glucose solution is facilitated, like that of Ringer and of physiological saline, by the rhythmic intestinal movements but the time taken is somewhat longer. Tenth normal hydrochloric acid causes a marked local contraction which prevents the passage of fluid even under comparatively high pressure.

OBSERVATIONS ON THE LIFE-CYCLE OF PLEUROPNEUMONIA VIRUS. K. B. Merling-Eisenberg. Brit. Jour. Exp. Path., xvi (1935), 4, p. 411.

Evidence is given to demonstrate a life-cycle for the virus of pleuropneumonia as follows: (1) elementary discs, (2) elementary discs, (3) discs with peripheral elementary bodies, (4) spheres with organization of elementary bodies inside, (5) spheres breaking up and thus releasing elementary bodies.

RABBIT POX. Susceptibility as a function of constitutional factors. Harry S. N. Greene. Jour. Exp. Med., lxii (1935), 3, p. 305.

The epidemiological significance of age, sex, genetic constitution and physiological status were studied by means of a differential analysis of the mortality data derived from a devastating epidemic of rabbit pox and, with the exception of sex, all were found to be factors of the utmost importance in the determination of susceptibility. Young animals were more susceptible than adults. Increased susceptibility was incident to lactation. Racial variations in susceptibility were of importance in adult populations. Proximity of relationship increased susceptibility. The final susceptibility or resistance was the result of the combined interaction of essential racial characters and the constitutional factors incorporated in the stock.

RABBIT POX. Paul D. Rosahn and Ch'uan-K'Uei Hu. Jour. Exp. Med., lxii (1935), 3, p. 331.

The clinical manifestations of rabbit pox consist of a generalized papular eruption involving the skin and mucous membrane together with blepharitis, ophthalmia, nasal discharge and lymphadenopathy. Pathological findings consist of vacuoliza-

tion, local necrosis and vesical formation in the epidermis while microscopic lesions may be confined to the corium. Infection can be transmitted through the medium of a personal carrier and such transmission can occur during the incubation period or before a definite diagnosis is possible. The etiological agents responsible for two outbreaks of rabbit pox were immunologically related. Immunity in recovered animals effectively persisted for nine to twelve months of available data. Young suckling animals of immune does were more refractory to the development of lesions of rabbit pox than were the young of the susceptible does.

PHENOMENON OF LOCAL SKIN REACTIVITY TO BACTERIAL FILTRATES IN RELATION TO ROUS CHICKEN SARCOMA ANTIBODIES. Gregory Shwartzman. Jour. Inf. Dis., lvii (1935), 2, p. 129.

Under the conditions of the experiment the phenomenon of local skin reactivity to bacterial filtrates cannot serve as an indicator of anti-Rous sarcoma antibodies. Rous sarcoma extracts and filtrates thereof do not contain the skin preparatory or reacting factors necessary for the elicitation of local skin reactivity.

TRANSMISSION OF EQUINE ENCEPHALOMYELITIS BY MOSQUITOES. Carl Ten Broeck. Abst. Amer. Jour. Path., xi (1935), p. 831.

Aedes sollicitans, the salt marsh mosquito found most abundantly in the regions in the East where the disease occurs, can be infected by feeding on infected brain mixed with blood or an infected guinea pig or horse. It apparently retains the virus as long as it lives and regularly transmits it by biting. Transmission of the virus from infected to normal guinea pigs and from infected horses to normal guinea pigs and a horse has been obtained. *Aedes cantator, Aedes taeniorhynchus* and *Aedes vexans* will also act as transmitting agents. Transmission experiments using *Culex pipiens* and *Anopheles quadrimaculatus* have been uniformly negative. In making transmission experiments mosquitoes should be fed material containing virus of high titre.

A STUDY OF THE PALATABILITY AND POSSIBLE TOXICITY OF ELEVEN SPECIES OF CROTALARIA ESPECIALLY OF C. SPECTABILIS ROTH. R. B. Becker, W. M. Neal, P. T. Dix Arnold and A. L. Shealy. Jour. Agr. Res., 1 (1935), 11, p. 911.

Grazing and feeding trials indicated that at least eight out of ten introduced species of Crotalaria are probably not toxic for

cattle. *C. retusa* were not grazed. *C. spectabilis* was definitely toxic to cattle. Bloody feces, hemataxia, loss of appetite, and general depression were the clinical symptoms observed. Petechiae were present in the mesenteric fat, gall-bladder and serous membranes of the viscera. Ecchymoses were observed in the heart, frontal sinus and trachea. *C. spectabilis* was least palatable of the species fed. *C. anagyroides, C. lanceolata, C. maxillaris, C. striata* and *C. usaramoensis* appeared not to be toxic. *C. intermedia* appeared to be the most palatable.

AN INHERITED SKIN DEFECT IN CATTLE. W. M. Regan, S. W. Mead and P. W. Gregory. Jour. Hered., xxvi (1935), 9, p. 357.

A recessive, sub-lethal epithelial defect which was uncovered in an inbreeding experiment is reported in Jersey cattle. The defect seems to be identical with one reported by Hadley in Holsteins. Parts of the body are hairless; there were no-skin lesions below the knees or hocks, around the eyes or on the muzzle. In some cases skin is present in patches below the hips. Local areas in other parts of the body are devoid of skin. The ears may be deformed, forming adhesions with the epithelium covering the head. In one female the anus was closed while in another there was no vulva. A breeding program is outlined to eliminate the lethal defect in Jerseys.

PUBLICATIONS RECEIVED

The Position of the Veterinary Surgeon in the Control of Milk, with Special Reference to Communicable Diseases. P. F. Dolan. Paper presented at 53rd Annual Congress, N. V. M. A. of Great Britain and Ireland, July 29-Aug. 2, 1935. pp. 9.

Notes on the Commoner Helminth Parasites of the Respiratory and Alimentary Tracts of the Domestic Ruminants. Col. W. A. Wood. Paper presented at 53rd Annual Congress, N. V. M. A. of Great Britain and Ireland, July 29-Aug. 2, 1935. pp. 10.

The Work of Veterinary Officers, with Special Reference to the Production and Inspection of Imported Meat Foods. Lt. Col. T. Dunlop Young. Paper presented at 53rd Annual Congress, N. V. M. A. of Great Britain and Ireland, July 29-Aug. 2, 1935. pp. 7.

The Modern School of Thought on the Practical Effect of Certain Unsoundnesses on the Serviceability of Riding Horses. Major C. H. S. Townsend. Paper presented at 53rd Annual Congress, N. V. M. A. of Great Britain and Ireland, July 29-Aug. 2, 1935. pp. 7.

Liberty exists in proportion to wholesome restraint.—DANIEL WEBSTER.

Regular Army

Major Raymond Randall is relieved from assignment and duty at the Boston quartermaster depot, Boston, Mass., and will report to the commanding general, First Corps Area, Boston, Mass., for duty with the Veterinary Corps at his headquarters and for additional duty at the Boston quartermaster depot and as attending veterinarian at Harvard University, Cambridge, Mass.

Major Nathan M. Neate, San Francisco, Calif., will proceed to Walter Reed General Hospital, Army Medical Center, Wash., D. C., and report to the commanding officer of that hospital for observation and treatment.

By direction of the President, Colonel Burt English, upon his own application, is retired from active service, to take effect October 31, 1935, under the provisions of section 1243, Revised Statutes, after more than 32 years of service.

1st Lieut. Daniel S. Stevenson, now on temporary duty at Fort Hoyle, Md., is relieved from further assignment to station at Fort Bliss, Texas, and will report to the commanding general, Fort Hoyle, Md., for duty.

So much of par. 30, S.O. 219, W.D., 1935, as relieves Captain Ralph W. Mohri from his present assignment and duty at Fort Riley, Kans., and directs him to proceed to New York, N. Y., and sail on or about December 31, 1935, for the Philippine Department, is revoked.

1st Lieut. Ray S. Hunsberger is relieved from his present assignment and duty at the Presidio of Monterey, Calif., effective in time to proceed to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about January 21, 1936, for the Philippine Department, for duty.

The appointment of the following-named second lieutenants, Veterinary Corps Reserve, as first lieutenants in the Veterinary Corps, Regular Army, with rank from September 30, 1935, is announced. Each officer named is assigned to the station specified opposite his name and will proceed from the place or station indicated to the station to which assigned and report to the commanding officer for duty accordingly:

Velmer Wayne McGinnis, Fort Des Moines, Ia.—Fort Des Moines, Iowa.

John Howard Rust III, Fort Williams, Me.—Fort Ethan Allen, Vt.

Bernard Francis Trum, Fort Adams, R. I.—U. S. Military Academy, West Point, N. Y.

Lloyd Christopher Tekse, Glencoe, Minn.—Fort Snelling, Minn.

Duane LeRoy Cady, Arlington, Nebr.—Fort Riley, Kans.

Edwin Louis Millenbruck, Fort Moultrie, S. C.—Fort Bragg, N. C.

Thomas Carlyle Jones, Fort Geo. Wright, Wash.—Fort Geo. Wright, Wash.

Major Samuel G. Kielsmeier is relieved from duty at the Robinson quartermaster depot, Fort Robinson, Nebr., effective on or about Jan-

uary 1, 1936, and will then proceed to Fort Ethan Allen, Vt., and report for duty.

Captain Maurice W. Hale is relieved from assignment and duty at the Army Veterinary School, Army Medical Center, Washington, D. C., effective on or about January 1, 1936, and will then proceed to Fort Sam Houston, Texas, and report for duty.

Major John H. Kintner is relieved from further assignment and duty at Fort Sam Houston, Texas, effective on or about January 10, 1936, is then assigned to the San Francisco port of embarkation, Fort Mason, Calif., and will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for temporary duty until such time as will enable him to comply with this order; will then proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about March 13, 1936, for San Francisco, Calif.; upon arrival in San Francisco will report to the commanding general, San Francisco port of embarkation, for duty.

The resignation by 1st Lieut. Duane LeRoy Cady of his commission as an officer of the Army is accepted by the President.

1st Lieut. Thomas C. Jones is relieved from duty at Fort Geo. Wright, Wash., effective on or about November 1, 1935, will then proceed to the Presidio of Monterey, Calif., and report for duty.

So much of par. 31, S.O. 219, W.D., 1935, as assigns Capt. Arvo T. Thompson to duty at Fort Riley, Kans., is amended so as to assign him to duty at the Army Medical Center, Washington, D. C., upon completion of his present tour of foreign service.

Veterinary Reserve Corps

PROMOTIONS

To

Milman, Maurice Howard.....Capt...240 W. 72nd St., New York, N. Y.
Granholm, Paul Robert.....1st Lt...Care Jones Dairy Farm, Inc., Fort Atkinson. Wisc.

NEW ACCEPTANCES

Johnston, Clarence Barney..2nd Lt...326 P. O. Bldg., Baton Rouge, La.
Krill, Walter Roland.....2nd Lt...Vet. Clinic, Ohio State Univ., Columbus, O.
Maher, Martin Patrick.....2nd Lt...Cromwell, Minn.
Scott, John Russell.....2nd Lt...309 5th Ave., So., South St. Paul, Minn.

Raising Ground Hogs Her Specialty

"Blackie," a 7-year-old dog owned by William Robertson, west of Washington, Iowa, this year mothered three ground hogs and a fawn, according to the *Cedar Rapids (Iowa) Gazette.* Blackie weaned her strange litter recently. Never a mother herself, Blackie likes the rôle of foster mother. This is the second litter of ground hogs she has nursed in the last few years. Arrangements have been made with Mr. Robertson to have Blackie and her "family" at the Iowa State Fair.

"CURES" AND CURE

By PHILIP P. JACOBS

Fifty years ago last May, an unattractive little red building with one room was perched on a hillside overlooking the Saranac River on the outskirts of what is now the village of Saranac Lake. To this cottage came two working girls, the first of a long line of thousands of tuberculosis patients who have been treated for tuberculosis at the now world famous Trudeau Sanatorium.

Dr. Edward Livingston Trudeau, himself a victim of tuberculosis, practiced the cure of this disease in his own life and from his own experience and that of a few other men, particularly two prominent German physicians, Brehmer and Dettweiler, he developed a regimen of living which has become the standard cure for tuberculosis. Out of that little red cottage have gone influences and technics that have revolutionized the health of the United States. When that cottage was first opened in 1885, nearly 300 people out of every 100,000 were dying of tuberculosis. Today less than 60 people out of every 100,000 are dying from this disease in the United States. The cure for tuberculosis that Trudeau developed, and which has been perfected along the lines that he started, is simply a way of living that comprises three essentials, rest in large doses, fresh air, and good food. After fifty years, we still cure tuberculosis with the same trio.

To be sure there have not been wanting men and women who have ventured to try this, that and the other thing in order to short-cut nature's way of curing tuberculosis. Almost every known substance, mineral and vegetable, organic and inert, has been tried singly and in combination as a cure. Hundreds of attempts have been made to kill the tubercle bacillus, the deadly germ of this disease, in the tissues of the body, but every at-

tempt has resulted in failure. For the substance that would kill the living germ in the tissues also kills the tissues—the cure is much worse than the disease. Even salts of gold or sanocrysin, one of the latest remedies to be tried, has proved a failure, as have salts of all the baser and precious metals.

The germ of tuberculosis, once it lodges in the body, is well nigh impregnable to any chemical or similar substance now known to man. Honest scientific investigators who have tried and failed deserve great credit for their endeavors. To those quacks who have exploited the consumptive public with worthless nostrums and have preyed upon their weaknesses, no words of condemnation are strong enough.

The National Tuberculosis Association has in its files records of more than 1,000 different kinds of "cures" for tuberculosis ranging all the way from such perfectly harmless things as lemons, or possibly dog's blood, to the most absurd contraptions and devices that one could possibly think of. Every kind of inhalant or every kind of drug or combination that could be thought of has been tried, all without success. "Cures," or as the doctor calls them, "specifics" for the treatment of tuberculosis, are of no value. The only known cure for tuberculosis is still Trudeau's remedy of fifty years ago—rest, fresh air and good food.

Today, as we carry on the work of the founder of the tuberculosis campaign in the United States and with the annual Christ-

MEDICAL IMMORTALS

Successful progress in the fight against tuberculosis is made possible chiefly by the discoveries of these three men. Robert Koch (left) noted German research worker, discovered the tubercle bacillus and proved it was the cause of tuberculosis in 1882. Rene Theophile Hyacinthe Laennec (center) young French medical genius, invented the stethoscope in 1815. When only 45 he became a victim of the disease he did so much to help conquer. Wilhelm Konrad Roentgen (right) noted German physicist, discovered in 1885 what is probably the most important diagnostic aid in modern medicine—the X-ray.

mas Seal sale promote a rational attitude toward the cure and prevention of tuberculosis, we are giving a new lease on life to thousands of men and women and we are hastening the day when tuberculosis will be brought completely under control.

Doctor Donham Joins Ohio State

Dr. Charles R. Donham, who was a member of the staff of the Veterinary Division of the University of Minnesota for six years, has resigned to accept the position of Professor of Veterinary Medicine at Ohio State University. Before going to Minnesota, Dr. Donham was connected with the Department of Veterinary Medicine of the Oregon State Agricultural College for seven years.

DR. C. R. DONHAM

Following his graduation from Iowa State College in 1921, Dr. Donham spent the summer of that year as veterinarian to the Saint Louis National Stock Yards Company. Then, after a year in private practice, he accepted a position as instructor at the Corvallis, Oregon, institution. He was promoted to assistant professor three years later. It was while Dr. Donham was located in Oregon that he became interested in the so-called salmon poisoning of dogs. Several articles on this subject have been published in the JOURNAL. While at Minnesota, from 1929 until the present year, Dr. Donham was engaged largely in laboratory investigations of Bang's disease. His name has appeared in

connection with numerous papers on that subject as well as others published in the JOURNAL during recent years.

Dr. Donham holds memberships in Phi Kappa Phi, Gamma Sigma Delta and Sigma Xi. His addition to the veterinary teaching staff at Ohio State comes at an opportune time, as Dean Brumley's staff has been very much overworked in handling the large increase in veterinary students during recent years. Minnesota's loss is Ohio's gain.

Minnesotans Honor Doctor Cotton

Dr. Chas. E. Cotton, secretary and executive officer of the Minnesota State Live Stock Sanitary Board, was the guest of honor at the annual banquet of the Minnesota Public Health Association, held in the ballroom of the Nicollet Hotel, Minneapolis, the evening of November 22, 1935. The banquet was attended by about 400 persons, including representatives of various organizations interested in public health. About 50 veterinarians and their wives were among those present.

DR. CHAS. E. COTTON

Dr. Chas. H. Mayo, of Rochester, president of the Minnesota Public Health Association, presided, and in presenting Dr. Cotton with a life membership in the association, Dr. Mayo paid a very nice tribute to Dr. Cotton and the veterinary profession of Minnesota. Other speakers included Dr. Morris Fishbein, editor of the *Journal of the American Medical Association*, Mr. F. E.

Murphy, publisher of the *Minneapolis Tribune*, and Dr. J. A. Myers, professor of preventive medicine, University of Minnesota.

Dr. Cotton has been identified with public health and live stock sanitary control work in Minnesota since 1895, and this recognition, by the medical profession, of 30 years of useful service in these fields, is very gratifying to members of the veterinary profession. Minnesota has always been regarded as one of the states having strictly modern methods for the control of animal diseases, based on a forward-looking plan.

Minnesota
Public Health Association

In Recognition of Distinguished Service in the Fight Against Tuberculosis, the Executive Committee Confers a

LIFE MEMBERSHIP

In the Minnesota Public Health Association upon

C. E. Cotton

Pioneer Leader in the Campaign to Eradicate Tuberculosis in Cattle
Public Health Statesman and Educator
Contributor to the Protection of Humanity

C. H. MAYO, PRESIDENT J. A. MEYERS, CHAIRMAN
E. A. MEYERDING, EXECUTIVE SECRETARY

CERTIFICATE PRESENTED DOCTOR COTTON

SOUTHWESTERN MINNESOTA VETERINARY MEDICAL ASSOCIATION

The regular semi-annual meeting of the Southwestern Minnesota Veterinary Medical Association was held at the Jackson Armory, Jackson, September 19, 1935. About 80 veterinarians were in attendance. The meeting was called to order by Dr. E. R. Tillisch, of Westbrook, at 1:00 p. m. and the following papers were presented:

"Diseases of Cattle," by Dr. W. L. Boyd, of the University of Minnesota, Saint Paul.

"Animal Disease Control and Community Sales," by Dr. C. E. Cotton, Minnesota State Live Stock Sanitary Board, Saint Paul.

"Enteritis in Swine," by Dr. J. D. Ray, Omaha, Nebraska.

Dr. T. W. Munce, of Sioux City, Iowa, led the discussion of the paper on enteritis. A banquet was served at the Ashley Hotel following the afternoon session and Redwood Falls was chosen as the place for the next meeting, which will be held in April 1936.

L. E. STANTON, *Secretary.*

MAINE VETERINARY MEDICAL ASSOCIATION

The regular quarterly meeting of the Maine Veterinary Medical Association was held at the DeWitt Hotel, Lewiston, October 9, 1935. The speaker of the evening was Dr. S. F. Griesemer, U. S. Bureau of Animal Industry inspector, stationed at Auburn, Maine. Dr. Griesemer covered the subject of meat inspection in all of its phases, as observed by him during his experience in this work for the federal government.

R. E. LIBBY, *Secretary.*

CHICAGO VETERINARY MEDICAL ASSOCIATION

The regular monthly meeting of the Chicago Veterinary Medical Association was held at the Palmer House, Chicago, October

8, 1935, with a large attendance of veterinarians from Chicago and vicinity. Dr. R. L. Tinkham presided.

Dr. S. H. Regenos, of Pitman-Moore Co., Indianapolis, Ind., gave a very interesting talk on "Laboratory Diagnostic Methods in Veterinary Practice." Mr. Floyd Young, superintendent of the Lincoln Park Zoo, of Chicago, related some of his experiences under the title, "Care of Animals in the Zoo."

Drs. H. W. Leib and C. C. Hastings, president and secretary, respectively, of the Illinois State Veterinary Medical Association, were in attendance and outlined some of the plans for the next annual meeting of the Association, which will be held in Chicago, probably in February.

Dr. L. A. Merillat spoke on "The Veterinary Service of the United States."

O. NORLING-CHRISTENSEN, *Secretary.*

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The semi-annual meeting of the Michigan-Ohio Veterinary Medical Association was held in the Masonic Temple at Blissfield, Mich., October 25, 1935, with 26 veterinarians in attendance. The program was begun at 4:30 o'clock with a talk by Dr. A. J. Kline, of Wauseon, Ohio, on the castration of cryptorchid horses. He was followed by Dr. Harry A. Hoopes, of La Rue, Ohio, who talked on his 20 years of experience with commercial dog-kennels.

Supper was served at 6 o'clock, and Mr. Elmer A. Beamer, of Blissfield, a real dirt farmer, was the first speaker. Mr. Beamer is president of the Michigan Live Stock Exchange, and he gave a very excellent address pertaining to the future of agriculture in the United States as it may affect the veterinary profession.

Dr. B. J. Killham, extension specialist, Michigan State College, a charter member of the Association, added to the success of the meeting by his talk on the W. K. Kellogg Foundation community health program. He outlined the work which the Kellogg Foundation has planned in connection with community health and pointed out the part to be taken by veterinarians.

As the regular election of officers was not held at the annual meeting in the spring, officers were elected at this meeting, as follows: President, Dr. J. W. Marshall, Genoa, Ohio; vice-president, Dr. A. J. Kline, Wauseon, Ohio; secretary-treasurer, Dr. E. C. W. Schubel, Blissfield, Mich.

E. C. W. SCHUBEL, *Secretary.*

GEORGIA STATE VETERINARY ASSOCIATION

The twenty-ninth annual meeting of the Georgia State Veterinary Association was held at Albany, October 7-8, 1935. The attendance showed a good increase over that of the previous year, especially from the standpoint of the ladies present, and particularly in the large number of members present who had not attended the meetings of recent years.

The program was well balanced in that it attempted to cover the entire field of veterinary endeavor, from public health work to the problems of the men in the large cities and the rural districts as well. As the meeting was held in the live stock section of Georgia, most of the discussions related to horse, mule, cattle and swine practice. Rabies took up considerable time, however. It was the consensus that the single-dose vaccination against rabies is a valuable measure in the control of this disease among dogs.

The subject that attracted most attention was so-called "mad itch." There was some division of opinion as to whether this condition is a form of hemorrhagic septicemia, or a separate and distinct disease. A number of practitioners related personal experiences to show that the condition is a distinct entity and that in some cases, at least, the hog seems to be a carrier of the disease, as well as a victim. Cases were reported in mules, cattle and dogs. Usually the condition is referred to by the layman as "rubbing disease."

On the second day, a clinic for both small and large animals was held at the hospital of Dr. Hays. At the business session, the election of officers resulted as follows: President, Dr. Charles C. Rife, Atlanta; vice-president, Dr. R. D. Carr, Thomasville; secretary-treasurer, Dr. J. E. Severin, Atlanta (reëlected).

J. E. SEVERIN, *Secretary-Treasurer.*

EASTERN IOWA VETERINARY ASSOCIATION

The twenty-second annual meeting of the Eastern Iowa Veterinary Association, held at Cedar Rapids, October 15-16, 1935, was well attended and was one of the most successful in the history of the organization, over 200 practitioners from Iowa, Illinois, Wisconsin and Minnesota being in attendance.

President Iva Dunn, of Atkins, Iowa, in his opening address before a large audience, made numerous cogent recommendations for the advancement of the profession and the continued

activities of the Association. The report of Secretary-Treasurer J. J. Strandberg, of Belle Plaine, Iowa, showed the finances to be in a very healthy condition. The report of Dr. John B. Bryant, of Mount Vernon, Iowa, on the results of the operations conducted at the fifth annual clinic, in June, was very interesting. The remainder of the first forenoon was devoted to discussions of Bang's disease by Dr. S. H. McNutt, of Iowa State College, and Dr. A. L. Born, of Story City, Iowa.

During the afternoon session, equine practice was discussed very thoroughly under the leadership of Dr. G. E. Van Tuyl, of Paullina, Iowa, and Dr. George R. Fowler, of Iowa State College. Discussions of poultry practice, led by Dr. John R. Christian, of Woodhull, Ill., and Dr. Willis A. Beard, of Greenview, Ill., both practitioners, occupied the balance of the afternoon. The question-box, which concluded the program, was handled in an able manner by Dr. Frank Breed, of Lincoln, Neb.

At the annual banquet and ball, Dr. George R. Fowler proved to be a very entertaining toastmaster. He introduced Dr. J. C. Flynn, president of the American Veterinary Medical Association, and about a score of other prominent veterinarians, many of whom were from out of the state. Dr. Fowler also introduced Mr. Hal Trosky, star first baseman of the Cleveland Indians, American League baseball club, and son-in-law of a former president of the Association, Dr. J. C. Glenn, of Norway, Iowa.

The technical program for the second day began with a paper, "Dietetics of the Modern Canine," presented by Dr. Earl R. Kennedy, of Moline, Ill. The discussion which followed this paper was led by Dr. J. C. Flynn, pioneer small-animal practitioner. "Some Phases of Swine Disease Control Work," given by Dr. R. M. Hofferd, U. S. B. A. I. Veterinary Inspector, stationed at Cedar Rapids, captured the interest and inspired much discussion. "Diagnosis and Treatment of Clinical Cases at Iowa State College," by Dr. Fowler, again brought out the notebooks of the practitioners. "Feeding Diseases of Sheep," presented by Dr. L. D. Frederick, of Chicago, Chief Veterinarian for Swift & Company, was one of the most informative papers ever presented on this subject, and a great deal of discussion resulted.

The afternoon session was begun with a very practical and informative paper, "The Value of Manipulation in Equine Diagnosis," presented by Dr. W. A. Aitken, of Merrill, Iowa. This, and the closing paper, "Dairy Cattle Practice," by Dr. S. L. Stewart, of Olathe, Kan., held the attention of a large and interested audience. Another question-box, handled by Dr. Ashe

Lockhart, of Kansas City, ended the meeting, with a large audience voting for adjournment.

Officers for 1936 were elected as follows: President, Dr. Wm. S. O'Brien, Ryan, Iowa; vice-president, Dr. Jas. C. Carey, West Liberty, Iowa; secretary-treasurer (reëlected), Dr. J. J. Strandberg, Belle Plaine, Iowa. Dr. V. B. Vanderloo, Dubuque, Iowa, was reëlected a member of the Executive Council to serve with Dr. C. H. Waite, of Stanwood, Iowa, and Dr. R. E. Elson, of Vinton, Iowa.

JOHN J. STRANDBERG, *Secretary-Treasurer.*

INTERSTATE VETERINARY MEDICAL ASSOCIATION

Another splendid meeting, with a near-record attendance, was held when the members of the Interstate Veterinary Medical Association assembled under the able direction of Dr. D. C. Scott, of Tekamah, Nebr., in the Hotel Warrior, at Sioux City, Iowa, October 17-18, 1935. The official register showed the following states represented: Kansas, 1; Illinois, 2; Missouri, 6; Minnesota, 11; South Dakota, 21; Nebraska, 24; Iowa, 92; total, 157.

The "high spots" on the program started with the first speaker and ended with the last. It was an all-star cast, with each speaker having some vital message well presented and enthusiastically received. In spite of very interesting, lively discussions after each number, President Scott was able to keep the program moving along on schedule.

Dr. J. C. Flynn, of Kansas City, was first up on Thursday. He discussed "The Matron and Pups," giving many practical, as well as technical, pointers from his long experience as the first exclusive small-animal practitioner in the Middle West. Dr. H. C. H. Kernkamp, of the University of Minnesota, gave a very thorough discussion of that most perplexing of swine diseases—enteritis. His discussion of the terminology used for the etiological organisms involved was enlightening and was nicely associated with practical pointers for the control of the "enterites."

Next, Dr. L. D. Frederick, of Chicago, chief veterinarian for Swift and Company, to whom we are greatly indebted for his appearance, discussed "Diseases of Feeding Sheep." Dr. Frederick is really pioneering in this field and has much valuable technical information not available anywhere in our literature. Many were disappointed when his discussion was not available in form for distribution.

The program Friday morning was launched with a discussion of "Colt Shows as Practice Builders," by Dr. R. W. Hixson, of Falls City, Nebr. He reported a total of 239 horses and mules shown in their second annual community show this fall. The high spot for attendance and interest was reached when Dr. C. H. Covault (himself) discussed that most vexing of our present-day horse problems—encephalomyelitis. In his usual complete and masterful manner he simplified and clarified most of the details which have been puzzling the practitioners.

Next, Dr. E. L. Eggleston, of Alcester, S. Dak., spoke on "The Control of Equine Strongylosis," another of the tractor salesman's insidious friends. The discussion brought out many practical pointers, yet emphasized the crying need for more efficient vermifuges.

Dr. C. F. Schlotthauer, of Rochester, Minn., who was present as a courtesy of the Mayo Foundation, discussed three fowl diseases: gout and enterohepatitis in turkeys, and so-called "leukemias" of chickens. His points were splendidly illustrated by lantern-slides. Last, but not least, Dr. E. R. Truax, practitioner *par excellence* of Sac City, Iowa, gave a very practical paper on the diagnosis and treatment of many commonplace cattle diseases.

The banquet on Thursday evening drew a record crowd of 151. As toastmaster, President Scott introduced the pride of St. Paul, who is also the Iowa-Minnesota member of the Executive Board of the A. V. M. A., Dr. H. C. H. Kernkamp. "Kerny" presented the notables who were present and seven charter members of this Association, which was organized in 1914. The president of our national organization, Dr. J. C. Flynn, of Kansas City, responded with a timely message from the American Veterinary Medical Association. He noted that four members of the Executive Board were at the banquet.

Following the banquet, the Association was the guest of 13 veterinary commercial concerns at a splendid dance, which lasted into the small hours of the morning. The Association went on record as favoring an invitation to the A. V. M. A. to meet in Omaha in 1937.

The election of officers for 1936 brought Dr. E. L. Eggleston, of Alcester, S. Dak., to the chair, with Dr. E. R. Truax, of Sac City, Iowa, as vice-president, and Dr. W. A. Aitken, of Merrill, Iowa, again reëlected as secretary-treasurer.

W. A. AITKEN, *Secretary.*

PURDUE UNIVERSITY VETERINARY SHORT COURSE

The annual Purdue University Short Course for Veterinarians was held at the Veterinary Building, LaFayette, Ind., October 22-25, 1935. The first session began at 1:30 P. M. The pathology of hog diseases was discussed by Drs. D. D. Baker, J. F. Bullard and L. P. Doyle. Selected cases of cholera, colitis and influenza were autopsied and the lesions present described. A general discussion followed.

The program of the second day included a trip to the college farm and an inspection of the breeding flocks and feeder lambs. Prof. C. Harper discussed management of breeding flocks and rations for feeder lambs. Some time was given to a general discussion of disease problems. The group then returned to the Veterinary Building and the regular program was continued. A review of recent literature on poultry diseases, by Dr. L. P. Doyle, completed the morning program.

The afternoon session was of special interest. The following subjects were discussed: "Endocrinology," by Prof. C. M. James; "The Experimental Feeding of Corn Stalks and Corn Damaged by Ear Worms, Molds and Rot," by Prof. J. F. Trost and Dr. J. F. Bullard; "Calfhood Vaccination for Bang's Disease," by Dr. A. L. Delez, and "Encephalomyelitis," by Dr. M. S. Shahan, U. S. Bureau of Animal Industry. Dr. Shahan illustrated his discussion with two films showing field cases of the disease and cases produced by inoculation with virus.

Bang's disease was discussed at the Thursday morning session. The interest in Dr. J. L. Axby's paper resulted in the veterinarians asking Dr. Axby to send them mimeographed copies. Dr. H. Busman, in charge of the federal testing, discussed problems relating to the field work. Dr. W. G. Galloway, in charge of the testing laboratory, gave the veterinarians helpful advice in regard to the shipment of blood samples to the laboratory and filling out the test-charts. This was followed by a discussion of local and general anesthetics by Prof. C. J. Zufall, of the School of Pharmacy.

The afternoon session opened with an address by Dr. H. Preston Hoskins, who gave a very interesting discussion of the Oklahoma City meeting of the A. V. M. A. House of Representatives, and an analysis of present conditions in our veterinary colleges, with special reference to student enrollment. The dinner meeting was held in the Memorial Union Building. Following an address

by Dean J. H. Skinner, the toastmaster, Dr. H. Meade Hamilton, of Muncie, Ind., called on several veterinarians for short talks.

Dr. W. B. Craig, of Indianapolis, was in charge of the clinic. He was assisted by Drs. J. F. Bullard, C. C. Donelson, S. M. Friedley and G. F. Eichhorn.

R. A. CRAIG, *Reporter.*

NORTHWESTERN ILLINOIS VETERINARY MEDICAL ASSOCIATION

The seventeenth annual meeting of the Northwestern Illinois Veterinary Medical Association was held at the Freeport Hotel, Freeport, October 22, 1935, with about 50 veterinarians present.

The program was started off promptly at 1:30 o'clock, by Dr. Frank Thorp, Jr., of the University of Illinois, who showed a number of lantern-slides and discussed worm parasites of animals, including poultry.

Dr. Frank Breed, of Lincoln, Neb., gave a very interesting discussion on swine erysipelas, and later spoke on equine encephalomyelitis. His talks were deeply appreciated by all who heard them. Dr. L. E. Willey, of Sioux City, Iowa, spoke on swine diseases, and ironed out some of the difficulties of differential diagnosis.

Dr. James S. Healy, U. S. B. A. I. inspector in charge of Bang's disease control work in Wisconsin, spoke on this subject and gave much valuable information concerning how the disease is being handled in Wisconsin, the percentage of reactors being found, the percentage of reacting herds, the percentage of herds tested in the state, and the percentage of reactors found in subsequent tests.

Dr. H. Preston Hoskins, secretary of the American Veterinary Medical Association, gave some data on the enrollment of veterinary students in the various veterinary colleges this year, at the same time commenting on certain trends in veterinary education.

Dr. L. A. Merillat, of Chicago, discussed the veterinary service of the United States, as he sees it. He calls a spade a spade, so that no one goes away without knowing what the actual conditions are and what is needed to correct them.

Officers elected for the new year are: President, Dr. B. L. Lake, Rockford; vice-president, Dr. G. H. Norris, Pecatonica; and secretary-treasurer, Dr. Roy E. Kluck, Freeport, who was reëlected for his fourteenth term of office.

ROY E. KLUCK, *Secretary.*

MICHIGAN STATE COLLEGE SHORT COURSE IN MEAT AND MILK INSPECTION

The practicing veterinarians located in or near Allegan, Barry, Branch, Calhoun, Eaton, Hillsdale and Van Buren counties, Michigan, gathered at the Michigan State College, East Lansing, on the afternoon of Monday, October 28, 1935, for the opening of a special short course in milk and meat inspection. The short course, which continued through the week to Saturday noon, was conducted under the joint auspices of the Veterinary Division of the Michigan State College and the W. K. Kellogg Foundation Michigan Community Health Project, and was designed to better fit the practitioner for participation in the comprehensive Kellogg Foundation program. Each veterinarian who signified his willingness to attend the short course was presented with an honorarium.

The first session was in charge of Dean Giltner and was opened by him with appropriate introductory remarks. Dr. G. E. Totten, U. S. B. A. I. inspector-in-charge, Chicago, Ill., followed with a splendid paper on "The Federal Meat Inspection System." This fundamental subject evoked considerable discussion. Next, in logical sequence, there were discussions of problems encountered in municipal meat inspection, led by Drs. H. S. Atkins, E. E. Hamann and R. E. Hammond, in charge of meat inspection in Pontiac, Lansing and Flint, respectively.

The afternoon program was closed by Dr. C. H. Clark, State Veterinarian, whose subject was: "Location, Construction, Equipment and Management of Slaughter-Houses."

The initial speaker for the morning sessions, which were devoted to milk inspection subjects, was Dr. I. F. Huddleson, who ably presented the subject of "Brucella Infection of the Udder." Dr. E. J. McLachlan was next, with a very practical discussion of problems encountered in milk inspection. Dr. McLachlan, a veterinarian, is now in charge of the Health Department in Jackson, Michigan.

Dr. W. H. Haskell, of the U. S. Public Health Service, Chicago, who was the next speaker scheduled, did not appear until later in the week, when he gave a splendid address on "Milk Inspection and Public Health."

The meat inspection sessions continued with discourses on parasites and diseases important in meat inspection by Drs. L. B.

VETERINARIANS FROM SEVEN COUNTIES IN MICHIGAN ATTEND SHORT COURSE IN MILK AND MEAT INSPECTION AT EAST LANSING

Sholl and B. J. Killham. Slaughter procedures and the technic of meat inspection were demonstrated by Dr. C. F. Clark. The following afternoon, using the same material, Dr. Clark directed a laboratory study of lymphatics important in meat inspection.

Proceeding with milk inspection topics, Dr. E. D. Devereux discussed the methylene-blue reduction test and the standard plate count of milk. Later, the test and count were demonstrated. Dr. F. W. Fabian reported on an extensive milk ordinance survey, and Mr. Russell Palmer, Chief of Milk Inspection, Detroit, outlined methods of dairy farm inspection.

Just prior to a general discussion of the veterinarians' participation in the Kellogg Project, Dr. D. R. Coburn gave same interesting observations on adulterations and the use of preservatives in meat products. During the participation discussion, a pertinent committee report was presented and the veterinarians of the seven counties were divided into two groups and two sets of officers were elected. It is anticipated that these organizations will be important cogs in future educational work and participation plans.

The milk inspection program for the latter part of the week was crowded. Mr. C. S. Bryan discussed infectious mastitis and demonstrated the laboratory diagnosis of streptococcic mastitis; Dr. Devereux discussed and demonstrated the direct microscopic examination of milk; Mr. James Warner presented the views of the State Department of Agriculture with regard to milk inspection; Dr. E. F. Meyer, Chief, Food Inspection Division, Grand Rapids, told how to bring about the production of better milk; and Dr. George Taylor gave a two-hour demonstration of the physical examination of the udder.

The lectures on subjects pertaining to meat inspection were concluded by Prof. George Brown, who gave a very informative talk on "Methods of Curing and Preserving Meat," and Dr. C. F. Clark, whose discourse was entitled, "Inspection and Use of Meat Products and By-Products." There were two additional laboratory periods. One was directed by Dr. E. T. Hallman and was devoted to a study of pathological museum specimens, and another was a half-day spent at a nearby packing establishment.

A splendid, sustained interest was in evidence throughout the various sessions and the veterinarians in attendance were practically unanimous in declaring that much benefit had been derived from the short course.

B. J. KILLHAM, *Reporter.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, November 6, 1935. The speaker of the evening was Dr. Benjamin Schwartz, Acting Chief, Zoölogical Division, U. S. Bureau of Animal Industry, Washington, D. C. His topic was "Studies Made in Producing Immunity in Animals Against Internal Parasites." This proved to be a very interesting subject.

Dr. Schwartz showed how animals become self-cured and set up an immunity to a parasitic infestation. Then he explained how it had been possible to establish artificial immunity in rats against cat tapeworm. Animals which had been immunized did not develop liver cysts, while those which were unprotected showed heavy infestation.

It was also brought out by Dr. Schwartz how an animal becomes sensitized to the effects of parasitic infestations, but once they are freed of the condition and then are tested with extracts of a parasite, it is not uncommon for them to become allergic, due to oversensitivity. This phenomenon was described by Dr. Schwartz as an immunity reaction upon the part of the host. An animal is not a passive bag for parasites, but reacts against the destructive measures of each infesting parasite.

Numerous points of vast interest and importance were brought out by Dr. Schwartz, and it is expected that some day it will be possible, as a result of this work, to develop biological products with which we may protect animals against parasites.

R. S. MacKellar, Jr., *Secretary.*

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The annual meeting of the Hudson Valley Veterinary Medical Society was held at Poughkeepsie, N. Y., November 13, 1935, President Sheldon presiding.

A brief business session was held. Drs. Wright J. Smith and David B. Comstock were elected to honorary membership. Drs. F. H. Haner, F. W. Schutz and E. A. White were elected to membership. Dr. George L. Stringham, Wappingers Falls, N. Y., was elected President, and Major E. M. Curley, West Point, N. Y., Vice-President. The Secretary-Treasurer, Dr. J. G. Wills, Albany, N. Y., was reëlected.

Dr. C. E. DeCamp, of Scarsdale, N. Y., delivered an interesting paper entitled, "A Consideration of Urinary Calculi in Animals." This paper dealt with the subject quite specifically. The author pointed out that while the exact cause of calculi is unknown, infection is often an important complication. Removal of such infection is very important. Faulty metabolism may be a contributing factor. The use of a ketogenic diet, properly modified, may be helpful. An adequate supply of vitamins A and D are necessary in the relief and prevention of this formation. He stated that vitamin A should be of animal origin. The veterinary profession can aid in developing much needed information on this important subject by helping to collect additional clinical data from their observations.

After considerable discussion on this and other case reports, the meeting adjourned to meet in Albany, on February 12, 1935.

J. G. WILLS, *Secretary-Treasurer.*

SOUTHERN STATES VETERINARY MEDICAL ASSOCIATION

The twentieth annual meeting of the Southern States Veterinary Medical Association was held at the Piedmont Hotel, Atlanta, Ga., November 7-9, 1935. This was the first time the meeting extended over three days. The attendance was approximately 225, including 35 wives of veterinarians.

The literary program consisted of the following papers:

"Anthrax," Dr. B. M. Lyon, Pearl River, N. Y.
"Wound Treatment," Dr. H. B. Smith, Farmville, N. C.
"Swine Diseases," Dr. H. J. Shore, Fort Dodge, Iowa.
"Small-Animal Practice," Dr. J. C. Flynn, Kansas City, Mo.
"Recent Developments for the Veterinary Practitioner," Dr. E. J. Frick, Manhattan, Kan.
"The Use of Local Anesthesia in Large-Animal Practice," Dr. T. A. Sigler, Greencastle, Ind.
"Bovine Tuberculosis Eradication," Dr. A. E. Wight, Washington, D. C.
"The Live Stock Industry and Its Improvement in the South," H. R. Smith, Chicago, Ill.
"Meat and Dairy Inspection as Delegated to Reserve Veterinary Officers of the CCC," Col. Burton E. Seeley, V. C., U. S. A.

Dr. Sigler very ably demonstrated a motion-picture film on encephalomyelitis.

The entertainment the first evening consisted of a banquet, with Dr. M. Jacob, of Knoxville, Tenn., presiding as toastmaster. The principal speakers were Maj. Trammell Scott, civic leader and sportsman, and Dr. J. C. Flynn, president of the American Veterinary Medical Association.

The evening of the second day, a dance was given in the ballroom of the Piedmont Hotel. This brought forth the ladies in all their finery to dance the light and fantastic to a good southern orchestra. Further entertainment was provided for the ladies in the form of shopping and sight-seeing tours, a luncheon and theater parties.

On Saturday, a clinic was held at the hospital of Dr. J. L. Hopping. A large number attended and the material was ample. Dr. Sigler held the interest of the large-animal practitioners, and Dr. Flynn and Dr. Frick divided the honors in the small-animal clinic.

Officers were elected as follows: President, Dr. L. J. Kepp, Atlanta, Ga.; first vice-president, Dr. R. R. Sally, Orangeburg, S. C.; second vice-president, Dr. W. D. Hiscock, Orlando, Fla.; secretary-treasurer, Dr. M. R. Blackstock (reëlected), Spartanburg, S. C.

CHAS. C. RIFE, *Chairman of Program Committee.*

Memphis to Have Big Meeting

Veterinarians of Tennessee and Arkansas are planning a big meeting, to be held in Memphis, Tenn., January 20-21, 1936. The officers of the Tennessee Veterinary Medical Association have been working with the officers of the Arkansas Veterinary Medical Association in an effort to perfect plans for the joint meeting. Dr. John H. Gillmann, of Memphis, is chairman of a committee that is arranging the program and making the local arrangements.

Dr. J. C. Flynn, president of the American Veterinary Medical Association, has promised to attend the meeting, and two former presidents of the A. V. M. A. have indicated their willingness to assist with the program. They are Dr. A. T. Kinsley, of Kansas City, Mo., and Dr. T. A. Sigler of Greencastle, Ind. Others who will contribute to the program are, Dr. E. J. Frick, of Manhattan, Kan., and Dr. C. E. Salsbery, of Kansas City, Mo. Dr. Gillmann reports that practically every veterinarian within a radius of 100 miles of Memphis has promised to provide clinical material for the meeting.

In conjunction with the plans for the Memphis meeting, the officers of the Mississippi State Veterinary Medical Association have scheduled their annual meeting for January 23, in Meridian. It is expected that Drs. Flynn, Sigler and Frick will go from Memphis to Meridian for the purpose of helping with the program for the Mississippi meeting.

NECROLOGY

HENRY H. MYERS

Dr. Henry H. Myers, of Cleveland, Ohio, died at his home, January 18, 1935, of bronchial pneumonia following a heart attack a few weeks earlier.

Born at North Lima, Ohio, April 13, 1872, Dr. Myers received his early education in local schools. He then attended the Columbiana, Ohio, High School. His veterinary education was received at the Ontario Veterinary College. Following his graduation in 1895, he practiced at Tiffin and North Lima, Ohio, until the spring of 1906, when he entered the service of the U. S. Bureau of Animal Industry. His first assignment was meat inspection at Cincinnati. Then he was transferred to Cleveland, where he remained until his retirement in 1934, except for short assignments in connection with the outbreaks of foot-and-mouth disease in 1908 and 1914.

Dr. Myers joined the A. V. M. A. in 1918. He was a member of the National Association of B. A. I. Veterinarians. He is survived by his widow (née Pearl Cover), a daughter and three sons.

CHARLES G. BURTON

Dr. Charles G. Burton, of Clayton, Ind., died at his home, June 27, 1935. He had suffered a paralytic stroke eight years previously, from which he had never fully recovered. He had been confined to his home for about two weeks.

Born near Greencastle, Ind., November 1, 1865, Dr. Burton attended local schools, engaged in farming, and then conducted a bus line between Monrovia and Mooresville. In 1896, he located in Clayton and operated a livery barn there. Later he decided to study veterinary medicine and attended the Indiana Veterinary College. He was graduated in 1910 and thereafter conducted a very successful practice at Clayton.

He is survived by one daughter, one son, two brothers and two sisters.

EDWARD J. DRAKE

Dr. Edward J. Drake, of Pembina, N. Dak., died in a hospital in Winnipeg, Manitoba, May 30, 1935, about a week after he had entered the institution for an emergency operation.

At the time of his death, Dr. Drake was a member of the field inspection force of the U. S. Bureau of Animal Industry. He was a graduate of the Kansas City Veterinary College, class of 1906. He practiced in Seattle, Wash., for a time and then engaged in ranching at Toledo, that state. In 1914 he entered the B. A. I. service for the first time, but remained in the work for only a few months, returning to his ranch. In 1924 Dr. Drake again entered the B. A. I. service and at various times was assigned to field work at Pierre, S. Dak.; Houston, Tex.; Lincoln, Neb., and Seattle, Wash., before being ordered to North Dakota.

Dr. Drake joined the A. V. M. A. in 1912. He is survived by his widow, three daughters, three sons, a sister and a brother.

A. T. K.

GEORGE JOHNSTON BURTT

The death of Dr. George J. Burtt, on July 28, 1933, has just been reported. Death was due to diabetes.

Born at Centreville, New Brunswick, April 8, 1882, Dr. Burtt was graduated from the Ontario Veterinary College in 1912, and practiced in Centreville until 1914, when he removed to Fort Fairfield, Me. He practiced there until his death.

Dr. Burtt joined the A. V. M. A. in 1921. He is survived by his widow (née Pearl Wilson), three daughters and a son.

LOUIS H. MATHERS

Dr. Louis H. Mathers, of Sebastopol, Calif., died at his home August 11, 1935, following a protracted illness.

Born at Parkersburg, W. Va., Dr. Mathers attended local schools and then entered newspaper work in Washington, D. C., and later in New York City, where he became one of the first columnists writing for syndicate publications. His ambitions led him into other fields of endeavor, as time passed, and eventually he decided to study veterinary medicine. He was graduated from the Indiana Veterinary College with the class of 1903 and the following year he received a degree from the San Francisco Veterinary College. At one time, Dr. Mathers was veterinarian

for the P. T. Barnum shows and went west while acting in that capacity. In the interest of his health, Dr. Mathers decided to remain in California. Later he accepted a government position in the transport service and made several trips to the Orient and South Seas.

Dr. Mathers returned to San Francisco a short time after the earthquake and was engaged in relief work for a time. He then moved to Santa Rosa, Calif., where he engaged in practice, remaining there until 1910, when he moved to Sebastopol. During the World War, Dr. Mathers was commissioned as a second lieutenant in the Veterinary Corps, September 8, 1917, was ordered to active duty on June 6, 1918, and directed to report to Camp Greenleaf, Ga., for a course of instruction. He was assigned to duty at Camp Gordon, Ga., November 1, 1918, and discharged from service on March 8, 1919.

Dr. Mathers joined the A. V. M. A. in 1918. He was a member of the Masonic fraternity and the American Legion. He is survived by his widow, a son and a sister.

FREDERICK JOSEPH BURKEY

Dr. Frederick J. Burkey, of Houston, Texas, died at his home, October 14, 1935. He had been in ill health for some time. He was one of the oldest veterinarians practicing in Texas.

Born in Jonesboro, Ill., October 21, 1859, Dr. Burkey received his primary schooling in that place. Then his folks, who had migrated to the United States from Switzerland just before his birth, returned to Europe, where he received his education in preparatory schools. The family then returned to the United States and Dr. Burkey entered the Ontario Veterinary College, where he spent one year. He finished his course at the Chicago Veterinary College, receiving his degree in 1890. Later he studied medicine at the University of Texas, at Galveston, and received his M. D. in 1904. Dr. Burkey practiced veterinary medicine in Galveston for 14 years and then moved to Houston, where he continued in active practice until his death.

Dr. Burkey was a splendid character, a keen student and loved by all who knew him. His philanthropies were legion. He was one of the active workers and organizers in the erection of the County School for Girls and he participated in many similar types of charity. He was very active in Masonic circles and was a charter member of the Houston Veterinary Association. Every veterinary hospital in Houston was closed for two hours during

the funeral services as an honor to Dr. Burkey. He is survived by two daughters and a son.

J. G. H.

GEORGE I. BLANCHARD

Dr. George I. Blanchard of Kansas City, Kan., died at his home October 1, 1935, in his 70th year.

A native of Texas, Dr. Blanchard secured his veterinary education at the Kansas City Veterinary College. Following his graduation in 1911, he spent about a year in the service of the U. S. Bureau of Animal Industry. In July, 1912, with Dr. E. B. Hollecker (K. C. V. C. '11), he organized the Missouri Valley Serum Company, one of the first concerns to engage in the commercial production of anti-hog cholera serum. He was president of the company at the time of his death.

Dr. Blanchard joined the A. V. M. A. in 1917. He was a director of the Kaw Valley Bank and a member of Saint Peters branch of the Holy Name Society. He is survived by his widow and one son.

A. T. K.

WILLIAM HENRY HUDSON

Dr. Wm. H. Hudson, of Moultrie, Ga., died suddenly at his home on October 10, 1935, following a heart attack, at the age of 52. He was a graduate of the Cincinnati Veterinary College, class of 1910, and had been in the service of the U. S. Bureau of Animal Industry for more than 20 years. He had been assigned to meat inspection at Chicago, New York, Jacksonville, Fla., and Moultrie, Ga. He was a member of the National Association of B. A. I. Veterinarians. He is survived by his widow and two sons.

FREDERICK D. HALSEY

Dr. Frederick D. Halsey, of East Saint Louis, Ill., was killed in an automobile accident, November 17, 1935, three miles south of Belleville, Ill. A fractured skull was the cause of death. In the same accident, City Judge W. G. Mitchell was killed and four other persons were more or less seriously injured.

Born at Neosho, Mo., January 10, 1877, Dr. Halsey, a negro, was a graduate of Ohio State University, class of 1911, and had been in the employ of the U. S. Bureau of Animal Industry for about 23 years. He was well known among the negroes of East

Saint Louis, having been a leader in educational and civic enterprises. He was a member of the National Association of B. A. I. Veterinarians.

G. H. B.

RALPH B. HUNTER

Dr. Ralph B. Hunter, of Remsen, Iowa, died in Saint Mary's Hospital, Rochester, Minn., October 23, 1935, after a short stay in the hospital.

Born at Saltvale, N. Y., March 26, 1886, Dr. Hunter received his veterinary education at the Chicago Veterinary College. Following his graduation in 1917, he located at Cantana, Iowa. Then he entered the insurance business at Cherokee, Iowa. In 1921, he resumed practice, this time at Remsen, where he remained until his death.

Dr. Hunter joined the A. V. M. A. in 1918. He is survived by his widow, three sons and one daughter.

AUGUST M. HOUSER

Dr. August M. Houser, of Versailles, Ohio, died suddenly at his home, November 1, 1935. He had been suffering from heart trouble for several months.

Born at Piqua, Ohio, September 19, 1885, Dr. Houser attended local schools and then entered the Ontario Veterinary College. Following his graduation in 1908, he located at Versailles and had been in active practice there until his death.

Dr. Houser joined the A. V. M. A. in 1921. He was a member of the Ohio State Veterinary Medical Association. He took a prominent part in civic, fraternal and business affairs in his city. He was a director of both the Versailles Building Loan Company and the First National Bank of Versailles, and served as a member of the Versailles Board of Education. He was an Elk and a Mason. Dr. Houser is survived by his widow, two sons, a daughter and a sister.

CHARLES M. STULL

Dr. Charles M. Stull, of Columbus, Ohio, died at the National Military Home hospital, at Dayton, Ohio, on November 7, 1935. He had been in poor health for several years and had been taken to the hospital about a week before his death, following a stroke of paralysis.

Born near South Bend, Ind., January 11, 1867, Dr. Stull, a nephew of the famous Studebaker brothers of South Bend, attended local schools in that city and was a student at Notre Dame University for one year. He then decided to study veterinary medicine and entered the Chicago Veterinary College. Following his graduation, in 1892, he returned to South Bend to engage in practice. He was state veterinarian of Indiana, 1893-94. Later he became interested in the circus business. For a number of years he traveled with the Sells-Forepaugh show and later organized a circus of his own, under the name of the Stull Brothers Show.

During the World War, Dr. Stull was commissioned as a second lieutenant in the Veterinary Corps, July 5, 1917. He was directed to report to the Southern Department, July 10, and was assigned to duty with the Eighth Cavalry at Fort Bliss, Texas. He was also on duty with the Auxiliary Remount Depot at the same station. Later he was assigned to duty with the First Cavalry at Fort Douglas, Ariz., and Camp Bowie, Tex. He was promoted to first lieutenant, Dec. 11, 1917, and to captain, Aug. 2, 1918. He was discharged Dec. 10, the same year. While in service, Dr. Stull's health failed and he never fully recovered. Following the war, he lived at McConnellsville, Ohio, removing to Columbus in 1928.

Dr. Stull joined the A. V. M. A. in 1917. He was a member of the American Legion and of various Masonic bodies, including the Knights Templar. He is survived by his widow (née Mary Hulburt) and five children.

STEPHEN THOMAS FINNEGAN

Dr. S. T. Finnegan, of Chester, Ill., was found dead in bed at his home, November 12, 1935. He had been in ill health for a long time. Death was due to cirrhosis of the liver.

Born in Saint Louis, Mo., January 18, 1891, Dr. Finnegan was graduated from the McKillip Veterinary College in 1911. He also was a registered pharmacist and had practiced at Chester since 1915. He is survived by his widow, seven children and his mother.

JOHN P. SOMERS

Dr. John P. Somers, of Chicago, died November 13, 1935, at the home of his son, in Chicago, after a long illness.

Born in Ireland, April 30, 1859, Dr. Somers taught school in his native country until he came to this country in 1879. His education was obtained in the public schools of Dublin and at Saint Patrick College. Dr. Somers was graduated from the McKillip Veterinary College in 1907, and shortly thereafter entered the service of the U. S. Bureau of Animal Industry. He remained in the service until his retirement in 1930.

Dr. Somers joined the A. V. M. A. in 1917.

PERSONALS

MARRIAGE

DR. CAMERON W. ARGUE (O. S. U. '30), of Saskatchewan, Alberta, Can., to Miss Etta Helmer, of East Orange, N. J., at East Orange, September 30, 1935.

PERSONALS

DR. R. L. SCHMIDT (Ont. '31) has located at Port Washington, Wis.

DR. L. E. THOMPSON (Chi. '04) has removed from Alva, Okla., to Waxahachie, Texas.

DR. L. F. WORKMAN (McK. '14), of West Point, Ill., is building an office and garage.

DR. V. E. ISHEE (O. S. U. '35), of Huntsburg, Ohio, has entered practice at Middlefield, Ohio.

DR. W. F. BABB (O. S. U. '25), of Utica, Ohio, was elected mayor of his city at the November election.

DR. L. K. FLOWER (Mich. '19), of Delton, Mich., is building a hospital at the rear of his residence.

DR. E. S. DEUBLER (U. P. '05), of Narberth, Pa., is president of the Ayrshire Breeders' Association.

DR. J. C. MCNEILL (Ont. '05) has returned from Onaway, Mich., after being in Milford, Mich., for a short stay.

DR. L. P. MILLER (O. S. U. '34) has purchased a piece of property in Clyde, Ohio, and will erect a hospital on it.

DR. C. M. PRENTICE (O. S. U. '12) has resumed practice at Clyde, Ohio, after a three-year period of inactivity.

DR. E. J. HART (McK. '11) has been reappointed Livingston County (Ill.) Veterinarian by the Board of Supervisors.

DR. E. J. WATTERS (Chi. '07), of Houghton, Mich., is traveling in California. He was last heard from at San Jose.

DR. J. FRANK STEVENS (O. S. U. '97), of West Liberty, Ohio, sustained a fracture of one of his hips while vaccinating hogs recently.

DR. JAMES HARRISON (Ont. '90), formerly of Galien, Mich., and more recently of Gary, Ind., has removed to Buchanan, Mich.

DR. J. G. CATLETT (U. S. C. V. S. '16) is now back in Miami, Fla., after spending the summer at Elmont, Long Island, N. Y.

DR. CHAS. L. HAUPERT (O. S. U. '35), of Port Washington, Ohio, is taking postgraduate work in histology at Ohio State University.

DR. W. E. RASMUSSEN (Colo. '28) has changed his location from Kaysville, Utah, to Ogden, same state. Address: 3065 Ogden Ave.

DR. C. K. MINGLE (O. S. U. '30), of Columbus, Ohio, is taking post-graduate work in pathogenic bacteriology at Ohio State University.

DR. WM. J. CHANDLER (K. C. V. C. '09), who has practiced at Milton, Ill., for a number of years, is reported to have removed to New Berlin, Ill.

DR. ARTHUR QUINN (Chi. '10), of Sycamore, Ill., recently completed the remodeling of the building in which his office and garage are located.

DR. DAVID S. ELSASSER (O. S. U. '35), of Kenton, Ohio, is taking post-graduate work in pathological laboratory technic at Ohio State University.

DR. ALBERT SANDERS (Chi. '08), of Stephenson, Mich., is planning to build a hospital for small animals on property near his home, in the spring.

DR. F. H. RIESTER (Ind. '01) has returned to Buechel, Ky., after spending a month at Evansville, Ind., in the employ of the State Racing Commission.

DR. J. R. BROWN (O. S. U. '20), of Ottawa, Ill., has been reëmployed as La Salle County Veterinarian for another year by the County Board of Supervisors.

DR. F. P. WOOLF (A. P. I. '10), of Montgomery, Ala., is now connected with the Veterinary Department of Alabama Polytechnic Institute, at Auburn.

DR. DANIEL DE CAMP (K. S. C. '29) is now located at Oregon, Mo. He is engaged in poultry inspection for the U. S. Bureau of Agricultural Economics.

DR. J. T. BROWN (K. C. V. C. '15), of Belleville, Ill., has been reappointed Saint Clair County Veterinarian for another year, by the Board of Supervisors.

DR. HARRY E. HAPNER (Chi. '18), of Arcanum, Ohio, received several cuts and bruises when his car was hit by another car on Route 49, near Arcanum, on October 14.

DR. LESTER R. BARTO (U. P. '30), who spent the past year in post-graduate studies in Europe, is again associated with Dr. J. B. Engle (Corn. '26), at Summit, N. J.

DR. N. S. MAYO (Chi. '89), of Highland Park, Ill., accompanied by Mrs. Mayo, has gone to Florida for the winter. They will have their headquarters at Mount Dora.

DR. H. W. BROWN (Ind. '23), of Fort Wayne, Ind., addressed the G. E. Rod and Gun Club at its October meeting. His subject was "The Care and Training of Dogs."

DR. D. L. PROCTOR (O. S. U. '17), of Lexington, Ky., was the speaker at a luncheon meeting of the Thoroughbred Club of America recently. He spoke on "Bone Diseases of Horses."

DR. W. B. MASSIE (Mich. '16), of Boston, Ind., addressed the Hamilton (Ohio) Rotary Club, on October 17. He described the Rotary International convention, held in Mexico City, in June.

DR. NEAL MCNEAL (O. S. U. '11), who has practiced at Ansonia, Ohio, for the past 17 years, has arranged to take over the practice of the late Dr. A. M. Houser, at Versailles, Ohio.

DR. B. ROYER (Ont. '98), who has been with the Wisconsin Department of Agriculture and Markets for about ten years as assistant state veterinarian and supervisor of tuberculosis eradication, has asked for and been granted a leave of absence. Dr. Royer has returned to his old home at Shawano, Wis., where he plans to do a little private practice and take things easy for awhile.

JOURNAL

OF THE

American Veterinary Medical Association

FORMERLY

AMERICAN VETERINARY REVIEW

Original Official Organ U. S. Vet. Med. Ass'n.

Edited and Published for the

AMERICAN VETERINARY MEDICAL ASSOCIATION

By

H. PRESTON HOSKINS, CHICAGO, ILL.

Volume LXXXVIII

NEW SERIES VOLUME 41

January, 1936, to June, 1936

CHICAGO, ILL.

1936